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ENDOCRINOLOGIC ASPECTS OF SOME NEUROLOGIC CONDITIONS *

WALTER TIMME, M.D.

NEW YORK

Just forty years ago, in 1880, there appeared an article on myxedema in the *Boston Medical and Surgical Journal* by Dr. William A. Hammond. He reported the first case of the kind in America. Based on necropsies of similar cases in England, he came to the conclusion that the mental symptoms were due to the mucoid deposits throughout the brain structure and that probably the cause of the disease lay there. No mention or thought of thyroid disturbance entered. Since that time we have made some progress. Does any one doubt that today, confronting us, are states of disease in which we show the same lamentable lack of knowledge? In the same year, Dr. George M. Beard presented at the American Neurological Association a report on neurasthenia—nerve exhaustion—with remarks on treatment; Dr. Hammond presented a paper on thalamic epilepsy, and Dr. Gray a paper on the "Interconvertibility of Migraine and Epilepsy." The juxtaposition of these papers gave me a text for this evening's address, made forty years after their presentation. The only one of the affections cited, whose cause and treatment are specifically known today, is myxedema. My function this evening will be to apply to neurasthenia, migraine and epilepsy the results of observation and experience in these states from the point of view of endocrinology.

Do you remember the poor victims of Beard's "nerve exhaustion," with their fatigability, their constantly recurring complaints of arrhythmic heart, precordial pain and intestinal stasis; with the seemingly wild subjective descriptions of their constantly changing state; with no friend or physician to understand them or help them, and who, compelled to go their way alone and misunderstood, were finally left at the mercy of the "patent medicine" output of the country? We laughed at their complaints, scolded them, and in the meanwhile grew gray-haired. But we rarely helped them. Our effort was the one constantly repeated

* Read at the fortieth anniversary of the Boston Society of Psychiatry and Neurology, Jan. 15, 1920.

assurance—given with pill or tonic—"it is only a matter of a few years, and the thing will pass over leaving you as well or better than before." We deprived them of their coffee, their tea, their tobacco and their alcohol; we changed their surroundings and their habits, but we failed to change their religion. This they did themselves to remedy our neglect and became Christian scientists.

Do you remember the classic case of migraine that greeted us with our first patient of a busy day? He had been to ever so many physicians, each of whom had some new method for relieving him. Did you not inquire into his family history almost from the first and grasp with avidity the fact that his paternal grandmother had had the same kind of headache? And that the headache was relieved as soon as he had vomited, and that he really couldn't remember that any particular food was a necessary accessory before the fact, and that perhaps for a short while his vision had been disturbed, or his eyelids may have drooped? And a little more of this, and you were at the end of your tether, your questions came more slowly and finally stopped altogether. You maneuvered to find out what others had done for him, so that by no chance you would make a faux pas; then you made a pass in the dark—to no avail—and your scalp was added to those in his belt.

And we certainly have with us, even today, our constant companion, the patient with idiopathic epilepsy—unless, perchance, he has made newer friends among the abdominal surgeons, or the dentists or the psychologists. He was the bane of our calculations. He rarely had his attack when we thought he should; he would surprise us by freedom from attacks and give us credit for it, when we knew better; and bromid was his portion, however disguised, with an occasional treat in the way of crotalin, rattle-snake poison, or solanum carolinense. Shades of a benighted era! How low had we fallen!

But gentlemen, our attack on these enemies has taken another turn. Our tactics have changed. What we have heretofore regarded as entities are found to be merely incidents. And combined with numerous other incidents, though perhaps less manifest and hence requiring some search, a fairly completed disease picture can often be pieced together under each of the foregoing groups.

NEURASTHENIA

Our neurasthenic patient, according to our heretofore accepted examination, showed, neurologically speaking, perhaps a slight change in the reflexes, very little change from the normal in his urinalysis, and perhaps a slight anemia in the usual blood examination. His viscera seemed normal and even his blood pressure was not far wrong. We therefore told him there was no organic lesion, and perhaps reserved to ourselves the thought that his was some defense reaction

against his environment. Of a certainty, therefore, there was nothing to cure but his maladjustment. But not one of us is ever properly and entirely happily adjusted, and yet we do not all become neurasthenics. Why? If we had pursued our examination somewhat farther we would have found a number of curious facts, seemingly irrelevant, which repeated themselves in a majority of our cases. These facts I will endeavor to group successively. The first embraces the early history of our patient. He was late in cutting his teeth; he suffered from nose-bleed; he was a bed-wetter to the tenth year. A few years later he began to grow rapidly and began to have headaches. He had muscular cramps and spasms, "growing pains"; his arms looked like flails. His secondary hair—both on face and in the pubic region—came rather late. He was usually affected by cold weather, and his extremities were cold and damp. As his full growth was attained, these signs gradually disappeared and to all appearances he was well and sound. He married, was happy for some years, and then gradually he began to feel that he was somehow losing ground. He had a bronchitis, following which he began to lose vigor and initiative; he felt tired at slight exertion, his appetite began to wane, he lost weight. He knew that somewhere in his economy things were wrong; he felt his pulse, he sensed his heart throbbing in his arteries, he became so occupied with his search that any disturbing factor or even ordinary conversation made him irritable, and from that time on he began his neurasthenic stop-over journey around the medical world.

Now, however, we will give some facts of his present examination that are pertinent and that will explain many of his subjective complaints. Stripped, he will show disproportionate skeletal growth; arms too long for the body, a thorax too short for the legs or vice versa; teeth that are somewhat anomalous in that the lateral incisors are small and possibly decayed, the canines with attributes of incisors; hairy growth may disclose a feminine pubic distribution or other abnormal features. The scrotal fold may surround the base of the penis. The reaction of the skin to stroking will frequently, especially on the abdomen, show a white line, while on the thorax it will be pink. The blood pressure, if taken casually, may be found to be normal or even somewhat higher. But taken as it should be, namely, two or three successive systolic readings at minute intervals, a rapid systolic drop may be seen, the final reading being below normal. I have seen systolic readings taken in this way to be as different as 175: 130: 105: with a diastolic reading of 70. There is a general relaxation of the smooth muscle organs and of the tendons, so that flatfoot, sacro-iliac backache and visceroptosis are present. The laboratory report of the blood will show an almost normal red and white cell count and hemoglobin content. But if a more intensive examination is made, it will be found that there is

a decided drop in the polymorphonuclear count and a relative lymphocytosis; the carbon dioxid tension verges to the lowest normal, the sugar content is abnormal, the blood coagulability is delayed, and often there is an eosinophilia. Even with these few salient points in our examination, it will be seen that our attack is quite different from the old method. The interpretation of this picture is a faulty compensation of a suprarenal inefficiency. This suprarenal inefficiency has arisen as a result of some final causative factor, such as an infectious disease, but on a preformed basis of endocrine instability. The life history of the patient gives manifold evidences of such disturbances in growth and metabolism especially. Such suprarenal inefficiency may remain latent until the patient subjects himself to stress and strain beyond his narrowed limits of fatigue, or may be partially compensated for by other glandular structures, such as the pituitary. Overcome this compensation, and we have manifestations of neurasthenia, psychasthenia, and the psychoneuroses. Definite improvement may be obtained by appropriate therapy, based on each patient's individual incapacity for self-compensation. The very agents that heretofore were forbidden our patient—coffee, tea and alcohol—are extremely useful adjuvants in this treatment. The actual glandular therapy cannot be considered this evening.

MIGRAINE

Our attention is next drawn to our migrainous friend. If we dwell on his family history and antecedents, not only do we find attacks of similar nature, but several other points of significance. Many of the ancestors are tall—over 6 feet; some may have had diabetes; occasionally we find a goitrous person, and if the history is thoroughly looked into, many other endocrine anomalies may be disclosed. Again, during the early life of the patient, features of a disturbed endocrine balance are manifested, especially those of a dyspituitarism. Hair anomalies are frequent; disproportion in skeletal growth likewise; gonadal development is usually tardy. Girls menstruate rather late, and their periods usually are delayed. The actual migrainous attack quite frequently hinges about the menstrual period. Considering together some of the outstanding symptoms of a migrainous attack, we are struck by the fact that almost all can be accounted for by a disturbed pituitary body. There is a change in the blood pressure, which is usually quite low during the attack; a change in the blood sugar content; a retarding of the pulse rate, frequently down to 40 a minute; and at first a lowering of the temperature. The beginning of the attack with polyuria, the frilosity, the vasomotor spasms, all indicate pituitary involvement. The yawning which frequently precedes the attack is usually an accompaniment of fatigue on a basis of primary suprarenal and

secondary pituitary deficiency. If we examine the skull of our patient with the roentgen-ray, we will almost invariably find evidences of a small sella turcica, with erosions. The production of the migrainous attack is about as follows: At periodic intervals, such as menstrual recurrences, the pituitary body enlarges. Other factors producing such enlargements are fatigue, which, drawing out the available epinephrin supply, calls on the pituitary to compensate for the purpose of maintaining a proper blood sugar supply and a proper blood pressure; or intense cold, which, causing constriction of the superficial vessels, produces an enlargement of the deep lying organs, among them the pituitary; or intense mental occupation, which likewise produces an engorgement of the cerebral vessels, including the highly vascular hypophysis. Ingestion of certain kinds of food, notably carbohydrates in quantity, may also cause enlargement of the gland. If the sella turcica of a person subjected to these disturbing factors is too small to accommodate the enlarging gland, pressure against the surrounding structures of the hypophysis occurs. First, there is erosion of the surrounding bony capsule, and then pressure against the softer structures beyond. Such pressure occasions headache. This may be unilateral or bilateral, but is always at first referred to the intratemporal region. If the enlargement continues, the pressure begins to be exerted on either the cavernous sinus or anteriorly on the optic chiasm. But the cavernous sinus is in intimate relationship with the third, fourth and sixth cranial nerves, the ophthalmic division of the fifth with its lacrimal branch, and the sympathetic plexus of the carotid. Furthermore, it drains the ophthalmic vein. Is it merely a coincidence that migraine, as it progresses, involves practically all these structures, producing oculomotor palsies, disturbed lacrimation, hemianopsias, intra-ocular pain and pressure and perhaps exophthalmos, and finally, pressure on the carotid plexus, and universal headache? But you will say, if this is so, surely such conditions must occasionally be seen at necropsy. They have been seen. Deyl, at a Paris international congress a few years ago, reported several cases of migraine in which the postmortem examination showed a unilateral pressure exerted by the enlarged hypophysis on the cavernous sinus. I, myself, have among my roentgenograms, four that show a very marked unilateral erosion of the clinoids in patients suffering from migraine. About ten years ago, Plavec confirmed Deyl's findings. Hence in migraine, we have symptoms referable in the first place to metabolic disturbances caused by the pituitary, and secondly, to the direct pressure exerted mechanically by the enlarged gland. Whether those cases in which the headache begins in the occiput and which are occasionally accompanied by hemiparesis or hemianesthesia are of this same class, remains to be proved. For the present, it would be well to classify the migraines on some such

basis as I have indicated. It seems to me that this conception of the typical migraine attack with ocular symptoms is the rational one and explains practically the entire situation.

EPILEPSY

When we consider epilepsy, our problem changes somewhat. We must first classify our cases. Which are frankly endocrine, and which are not? Our criteria are found in the hereditary history, the developmental years and the present examinations. To give you all the endocrine stigmas to be encountered in these three groups would be to review the subject of endocrinology. Briefly, the grosser hereditary factors of importance are: abnormal skeletal growth, pronounced abnormal quantity and distribution of hair, obesity, diabetes, goiter and fatigue states; vasomotor disturbances, such as urticaria, Raynaud's disease, erythromelalgia and intermittent claudication. Pronounced cases of such complaints in the family must awaken the suspicion of an endocrinopathic heredity. The early life of the patient must show some of the endocrinopathic tissue changes or metabolic disorders for which we searched his heredity. There will be in his adolescence signs of fatigability, drowsiness, headaches; precocious or tardy genital development, invert signs of secondary sex characteristics, such as breasts in the male, mustache and male pubic hair in the female. In our examination of the patient, we must note each tissue, bone, muscle, skin, hair, nails and teeth. Is the jaw prognathous, are the teeth spaced or crowded, is the palate narrow and high or broad and shallow, is there much or little hair and is it properly distributed? Is the ratio of torso to leg normal; are the hands long and slender or broad and chubby? Are the skin reactions normal to stroking? Is the blood picture correct or have we a relative lymphocytosis, or an eosinophilia, or a low blood sugar content? Is the coagulation time between five and eight minutes, as it should be? What is the carbon dioxid tension? Blood pressure, temperature and pulse rate may also be suggestive. Is the thyroid large, or does the roentgen ray show any sella turcica abnormality? If our epileptic patient withstands to a great extent these inroads on his endocrine assets, then is he not of our present class. Those that fall well within our suspicions can be divided into two groups. The first is represented by the adipose, sluggish individual with smooth, velvety skin, body hairless, small genitals almost imbedded in the lower fatty abdominal folds, with a high sugar tolerance and a small closed-in sella turcica. This group represents the type of which Froelich's dystrophy is the classic. There are many variations with a corresponding change in the physical signs. Those of this group that have epileptic seizures—and there are many—frequently have the uncinate fit with aura of smell or taste. They

are distinctly pituitaric. Feeding them with appropriate doses of pituitary gland, frequently the anterior lobe extract only, produces marked improvement and even cures the disease. The epileptic fit is produced as a symptom in this state, much as is the migrainous attack mentioned in the foregoing. A periodic enlargement of the hypophysis, produced as before, in a sella not roomy enough to accommodate it, causes the critical moment. At times, in these cases, a migrainous attack accompanies the seizure, or is the equivalent of a seizure, and the uncinate features are presumably due to the extension of the pressure to the uncinate gyri of the temporosphenoidal lobe immediately surrounding the sella. This serves to explain the fact of the interconvertibility of migraine and epilepsy, cited forty years ago by Gray—an observation which in the experience of each one of us has had its examples.

The second group is represented by persons, likewise of endocrinopathic heredity and early life's history in whom the attack is secondary, in all probability, to retained nitrogenous products — presumably split protein and amino-acids. The retention in the tissues, in fact even in the individual cells, of these partial decomposition products, acts as an irritant which finally produces the epileptic seizure as a purposeful act; for the convulsion, accompanied by its profuse sweating, increased kidney and bowel action, tends to relieve this condition. The reason for the retention of amino-acids—partial protein decomposition products—in the cell, has been described ably by E. C. Kendall of the Mayo Foundation. He has shown that the iodothyroid product of the thyroid gland is instrumental in converting the amino-acids into ammonium carbonate, carbon dioxid and water, the ammonium carbonate being probably changed by parathyroid activity into urea. In these final forms, the protein combustion products are ready for excretion by the emunctories. Failure of the thyroid-parathyroid apparatus, then, means failure in the removal of the toxins. This situation determines the seizure. It is our task, therefore, to determine in which of our epileptic patients this condition obtains. I do not believe that there is a constant subthyroid state that is the goal of our quest. Such a state would determine a generalized hypothyroidism, a partial myxedema. A myxedematous state more or less is self-compensatory in that the person is sluggish, slow of mentality and activity, with a much lower basic metabolism than is normal. He burns less tissue, and there is less to excrete. But our dysthyroid epileptic is one in whom either periodically or after a period of excessive oxidation, the thyroid-parathyroid apparatus is insufficient. In other words, an instability of this glandular activity must be predicated. Such instability, however, is almost never primary, depending on its

covariants among the other endocrine organs for its production. A marked ovarian disturbance, a suprarenal outpouring, an underfunctioning pituitary — all may serve to make excessive demands on this thyroid-parathyroid organ, allowing insufficient secretion for the performance of its catalytic activity. This constellation of the units of the internal glandular system determines the attack. It is not a condition of pure hypothyroidism as it is pictured by some authors. Hence the stigmas of hypothyroid states are not successively found in these subjects. One may just as frequently find ovarian or suprarenal or pituitary characteristics. The main points to determine are the endocrinopathic family history and the presence of glandular disturbances in the early life of our patient. The treatment also would vary considerably depending on the result of our investigation into the primary disturbing element. If this could be found, and corrected, the seizures would cease. Frequently this can be done. Ovarian or gonadal disturbances are often amenable to treatment. Emotional states, fright, fatigue, anger, serve to call out epinephrin in too great amounts not to involve overactivity of the thyroid; but these conditions are difficult to combat. It is here that thyroid administration is indicated. Such administration should neither be continuous nor given in large dosage. Large doses would simply serve to increase the protein disintegration and would tend to increase the seizures. It is in these cases that proper administration reaches its highest art.

COMMENTS

I hope I have indicated in this comparatively short sketch, in which only the high lights could be pointed out, the bearing of endocrinology on some of our old problems of neurologic interest. You see, new points of attack are disclosed, and our patients are viewed, not as representing entities in their disease complexes, but as being simply the results of various combinations and permutations of constantly varying factors; their symptomatology, while manifestly widely differently constituted, has many basic similar ground plans. They must always be treated individually and as individuals, and never as group cases with definite limitations. Endocrinology, gentlemen, marks a new era in medicine.

MONOPLEGIA SPINALIS SPASTICA*

WILLIAM G. SPILLER, M. D.
Professor of Neurology, University of Pennsylvania.
PHILADELPHIA

We have had in the neurologic service of the University of Pennsylvania a number of patients in whom paralysis of one lower limb was associated with atrophy, exaggerated tendon reflexes of the affected limb and in some instances with spasticity. The first case of this kind was reported by Dr. Williams B. Cadwalader,[1] June, 1916, and is given as Case 2 of this paper. Since that time a considerable number of cases have been under observation, but it was not possible to devote sufficient time to scientific work during or immediately following the recent war. This form of monoplegia has also been studied by Sittig[2] and in all his cases the upper limb was affected. He had no necropsies. He suggests the term monoplegia spinalis spastica superior. In all my cases the lower limb has been the one affected, so that a lower as well as an upper type occurs, a monoplegia spinalis spastica inferior. It seems preferable therefore to describe the condition simply as monoplegia spinalis spastica, or in the English equivalent, spastic spinal monoplegia.

In some of Sittig's cases sensory disturbances were observed on the ulnar side of the paretic upper limb from a bullet wound of the cervical region. He found that the syndrome may result from different forms of spinal paralysis, from diplegia, hemiplegia or the Brown-Séquard complex, but it may be primary from the beginning. The condition differs from that in which there is hemiplegia from a spinal cord lesion in which the paralysis of the upper limb is merely more pronounced than that of the lower. He believed the explanation awaits further investigation. Fabritius came to the conclusion that the fibers for the upper limb are separate from those for the lower limb in the crossed pyramidal tract of the spinal cord, but Hoche, Fischer and Gierlich have shown by microscopic study that small lesions of the anterior central convolution of the cerebrum cause diffuse degeneration of the crossed pyramidal tract of the cord by the Marchi stain.

The most striking case, reported by Sittig is one in which spastic monoplegia of the left upper limb existed two years after a bullet wound of the neck had been received, while the other limbs had full

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* Read before the Philadelphia Neurological Society, Jan. 23, 1920.

1. Cadwalader, W. B.: J. Nerv. & Ment. Dis. **43**:547 (June) 1916.
2. Sittig: Monatschr. Psychiat. u. Neurol. **46**:112 (Aug.) 1919.

movement and sensation was not affected. Sittig had no pathologic findings to offer in explanation, but he believed there must have been a partial transverse lesion, probably an area of necrosis, in the upper cervical cord, and that it could not have been a lesion of the anterior horn, because of exaggeration of reflexes in both upper and lower limbs, especially in the upper, and because of absence of pronounced reaction of degeneration. The lesion could not be cerebral, as indicated by the course of the bullet and by the fact that in certain cases the symptoms must be of spinal origin. In one case the interossei muscles of the hand were atrophied and there was slight change of electrical reaction. There was not spasticity in all the cases Sittig reports. In Case 3 he distinctly says the tonus of the upper and lower limbs was normal; in some other cases of his series it was only a little exaggerated. In Case 7 both upper limbs were weak and the tonus of these limbs was normal. In Case 8 the tonus of the weak right upper limb was not increased.

There is some danger that spastic monoplegia of cortical origin from a lesion of the cerebrum may be mistaken for one of spinal origin, and this possibility should be kept in mind. The cerebral form has recently been the subject of a paper which I contributed to the anniversary volumes dedicated to Sir William Osler.[3]

Some of the cases of monoplegia with exaggeration of tendon reflexes result from injury of joint or bone in the affected limb, or from a condition which Babinski has called reflex paralysis. Where the bone or joint is affected there is likely to be pain, and spasticity is not conspicuous. Such a case is number 5 in my series, and it is reported because of its clinical resemblance to the other cases.

Even where the lesion is one of the spinal cord the spasticity may be slight, as in most of my cases, or pronounced as in my Case 7. The explanation which appeals to me for the spastic spinal monoplegia is one I[4] advanced in 1907. At a meeting of the Philadelphia Neurological Society on November 26 of that year I reported two clinical cases of poliomyelitis with exaggerated reflexes, gave references to a few similar cases in literature, and suggested an explanation for atrophy with spasticity and exaggerated reflexes of spinal origin on the supposition of a lesion resembling that of amyotrophic lateral sclerosis, i. e., with degeneration of both pyramidal tract and anterior horn of the spinal cord.

I described a case I had published in *Brain* in 1903, one of acute poliomyelitis with necropsy. The degeneration of the lumbar region

3. Spiller: Contributions to Medical and Biological Research Dedicated to Sir William Osler, J. Nerv. & Ment. Dis. **35**:258, 1908.

4. Spiller: J. Nerv. & Ment. Dis. **35**:261, 1908.

was typical of this disease and was confined to the anterior horns. Implication of the lateral columns occurred in a limited area in the thoracic region. The case offered an explanation for the exaggeration of tendon reflexes in poliomyelitis. It seemed to me that if the degeneration of the lower part of the cord does not implicate the cells innervating the thigh muscles, but is confined to those supplying the leg muscles below the knee, as is so often the case; and if the lateral column of one or of each side is implicated in the thoracic region, exaggeration of the patellar tendon reflex may occur. This point of view has received corroboration by a case of poliomyelitis with necropsy I have recently studied.

REPORT OF CASES

CASE 1.—*History.*—J. W., aged 62, in the service of Dr. C. K. Mills at the Philadelphia General Hospital, was admitted Dec. 31, 1917, and died Sept. 22, 1919. The right lower limb had been wasted since he was 2 years old. The limb was not only smaller but it was also shorter than the left. The statement was made that when he was 2 years of age he was taken outdoors by his sister, and when he was put down to walk his right lower limb gave way. Since that time the condition of this limb had been abnormal.

Examination.—The irides contracted sluggishly to light but promptly in accommodation. He had had a +++ Wassermann reaction. The grip of each hand was fairly strong. The right lower limb was slightly hypertonic. The triceps, biceps, patellar and Achilles' reflexes were all exaggerated on each side. No sensory changes were observed. The man was feeble in his limbs.

Necropsy Examination.—The lumbar and sacral roots could not be counted on account of the condition resulting from the necropsy. The lumbar and sacral regions were divided into blocks about 3/16 of an inch in thickness and sections were taken from each block. The upper part of the second lumbar segment appeared to be normal, but in the block immediately below this the greater part of the left anterior horn was degenerated and nerve cells and medullated nerve fibers had disappeared. The right anterior horn at this level was normal. The Marchi sections showed no distinct recent degeneration.

In sections from the next block the left anterior horn was normal, so that the degeneration of this side was limited. The right anterior horn showed a normal appearance as regards medullated nerve fibers but contained few nerve cells. From this level downward the degeneration of the right anterior horn in its outer portion was pronounced. In the upper levels the degeneration was confined to the outer portion, but a little lower the outer half of the right anterior horn was degenerated. In its inner half medullated nerve fibers could be seen, evidently coming from the left anterior horn through the anterior commissure. The degeneration of the right anterior horn extended from the middle of the lumbar region through the sacral region to its middle portion. Both crossed pyramidal tracts appeared slightly degenerated. A moderate degree of mononuclear cellular infiltration was found in the pia of the spinal cord and over the medulla oblongata, and this in connection with the Wassermann reaction and the sluggish iridic reflex to light suggested that the slight degeneration of the lateral columns probably was caused by syphilis. In addition, a small old area of softening was found in the right motor tract of the pons.

The exaggeration of the tendon reflexes probably was caused by the degeneration of the lateral columns, but as many of the cells of the right side of the cord in the outer half of the anterior horn of the lumbar region were destroyed and there were numerous medullated fibers in the inner portion of the right anterior horn in the lumbar region which came from the left anterior horn, it would appear that the exaggeration of the tendon reflexes in the right lower limb was partly dependent on these nerve fibers from the left anterior horn.

Case 2.—This case was presented by Dr. Williams B. Cadwalader in 1916 with the title "A Case Presenting Atrophy of the Right Lower Extremity with Increased Reflexes and Positive Babinski Sign."

History.—V. J. S., a boy, aged 17, was admitted to the University Hospital on Jan. 13, 1916, complaining of weakness and wasting of the right lower extremity. The patient's father was said to have had syphilis; his mother, two older brothers and two younger sisters were living and well. The patient stated that he had been well until 1913, when he had had an attack of what he had been told was rheumatism. This condition was characterized by pain on movement in the joints of both lower extremities and also in the right hand. There was no redness or swelling. After a few weeks he made a complete recovery. In September, 1914, he again had some pain in the region of his hip joints which was increased by movement. It disappeared, however, after a few days. He stated that since September, 1914, his right lower limb had been gradually growing weak and the muscles had been wasting. In all other respects he was entirely well.

Examination.—His eyes, cranial nerves, upper extremities and internal organs were found to be normal. The muscles of the right lower limb were moderately and uniformly atrophied and there was weakness in proportion to the wasting. The tendon reflexes were exaggerated, more so on the right side than on the left. There was a distinct Babinski sign on the right but not on the left side. Ankle clonus was absent. Sensation was normal. There was no incoordination, ataxia or tremor.

Examination of the joints by Dr. Edward Martin revealed nothing abnormal and in his opinion the atrophy was not related to joint disease. This was later confirmed by Dr. Pancoast who made roentgen-ray studies. Dr. Leopold made an electrical examination and reported that the muscles reacted normally to the faradic current. The Wassermann reaction was negative. Blood count and urine examination were also negative.

The combination of atrophy of gradual onset with increased reflexes and Babinski sign Dr. Cadwalader believed was to be explained by an infection which had caused pain in the joints simulating rheumatism, and had implicated the spinal cord as in acute anterior poliomyelitis. Some of the anterior horn cells probably had been destroyed and the pyramidal tracts had been involved.

Case 3.—*History.*—H. B., aged 8, was brought to the hospital by his mother, Feb. 12, 1919, because she had noticed that the left thigh was becoming smaller than its fellow. This was first noticed two months before. One year earlier the boy was run over by an automobile and his left leg was injured; he was obliged to remain in bed six weeks at that time. He said that he had not had any pain in the left hip or thigh, and his mother said he had not had any febrile condition during the past year. He had one brother and one sister, both living and well.

Examination.—The pupils, the ocular and other cranial nerves were normal. He had no signs of progressive muscular dystrophy. The entire thigh and leg

on the left side were smaller than the right lower limb, and the thigh was more affected than the leg. The patellar and Achilles' reflexes were present and exaggerated; the patellar reflex on the affected side was more exaggerated than the reflex on the other side. He had no Babinski sign and no ankle clonus. There was no change of sensation and no evidence of hip or vertebral disease.

Diagnosis in this case was difficult. The atrophy and exaggerated patellar reflex of one limb might permit the diagnosis of arthritic muscular atrophy, but as there had been no pain and there was no sign of joint or bone disease the possibility of a lesion of the spinal cord could not be excluded.

CASE 4.—*History.*—T. B., aged 19, was admitted April 25, 1919. There was no history of a similar disease in the family. He had always been well except for measles and mumps in childhood. He had no history of injury or disease before the atrophy was observed. He came to the hospital because of weakness and atrophy in the left lower limb. He noticed the weakness first in February, 1918, when he was doing endurance tests in the army, as he was a soldier. Soon after these tests he noticed that the left calf was smaller than the right.

Examination.—The circumference of the left calf was 2 inches less than that of the right. He flexed the left foot on the leg poorly, but had no difficulty in movements of the right foot. The left leg was markedly weak in resisting passive movement at the knee. The left patellar reflex was exaggerated but the left Achilles' reflex was not elicited. He had no clonus nor Babinski sign. The right tendon reflexes were normal.

While in France he carried about 70 pounds and did much hard marching over obstacles. At these times he had pain in the region of the left hip. The left sacro-iliac joint was photographed and was normal. The left foot was much smaller than the right, as if the bones had not developed.

The smaller size of the left foot suggested arrested development in childhood from poliomyelitis, especially as the left Achilles' reflex was absent. The severe strain caused by the endurance tests of the army may have caused the atrophy of the lower limb already inadequately innervated on account of the poliomyelitis. It is well recognized that atrophy may develop from old poliomyelitis, especially when the affected limb is put to excessive use. The exaggeration of the patellar reflex suggested further spinal disorder. There was no reason for a diagnosis of arthritic muscular atrophy, and yet there are certain superficial resemblances between this case and the following one in which the diagnosis of arthritic muscular atrophy was correct.

CASE 5.—*History.*—H. L., aged 25, was referred to me by Dr. Adlai S. Oliver of Benson, N. C., April 4, 1919. He had seen the patient only the day before he referred him.

The man was a soldier in France in the Engineering Corps when the symptoms began. He was obliged to take walks from 15 to 25 miles carrying from 70 to 80 pounds, and while doing this his right lower limb would give out. Symptoms began with pain in the right ankle in January, 1918, and not until six months later did he notice wasting and weakness of the limb. After about a month the pain seemed to be referred to the right hip joint.

Examination.—Examination at this time showed that the right calf was about ½ inch smaller than the left. A diagnosis of tumor of the spinal cord was made. Roentgen-ray plates of the spinal column and hip were negative. The Wassermann test, presumably of the blood, was repeatedly negative. He had no history of disease, either acute febrile or venereal.

The right lower limb was wasted. The thigh measured 2 inches less than the left in circumference, and the right leg 1 inch less. The whole right lower extremity was somewhat weak, but the man still had good use of the limb. The tendon reflexes were equally exaggerated in the lower limbs, but there was no ankle clonus. There was no Babinski sign and no disturbance of tactile, heat, cold, pain or deep pressure sensation in the lower limbs. There was no spasticity. He had become progressively worse.

An area of intense tenderness to slight pressure was found at the lower posterior and outer part of the right tibia. When examined by me the patellar reflexes were much exaggerated and equally so; the right Achilles' reflex was exaggerated, the left was about normal. The pain was constant in the right foot and usually increased by walking.

I strongly suspected some periosteal disease and sent him to Dr. Pancoast for roentgen-ray examination. He reported that there was an area of chronic localized osteomyelitis (infective type) at the lower end of the tibia. The lesion might easily be overlooked. He recommended operation.

CASE 6.—*History.*—F. C., aged 34, a carpenter, came to the hospital Jan. 26, 1920. His wife had had two children and no miscarriages. He had gonorrhea when 24 years old and had used alcohol moderately. Five years before he began to have pain in the right lower limb which at first was like a sensation as of pins and needles, and was felt chiefly in the anterior aspect and in the posterior part of the knee. He had no weakness but could not walk very well at that time because of the pain. He described the pain as a burning like fire, and it was worse at night. This pain lasted about a year. The right calf was operated on, and he had a large scar. The wound did not heal readily, and a year was necessary for recovery.

He had no more symptoms until ten days before he came to the hospital. He had been working as usual and one night when he came home from work he found he could not use the right lower limb in walking, and this was so weak he could not lift it. He had no pain and has not had any. He had no disturbance of the bladder.

Examination.—The right pupil was a little larger than the left but reaction was prompt. The face and upper limbs presented nothing abnormal. When he walked he had great difficulty in supporting himself on the right lower limb which he dragged along. It was smaller than the left, both in the thigh and calf muscles. He had no power in flexing the right foot on the leg, but had slight downward movement of the foot and was able to move the toes a little. He had little power in the right hip and knee, and when he uncrossed the right lower limb from the left knee the right limb fell to the floor. The patellar and Achilles' reflexes were exaggerated, and those on the right side were more exaggerated than those on the left side. Ankle clonus was persistent on the right side and abortive on the left side. Babinski reflex was typical on the right side and almost as typical on the left. He moved the left lower limb freely and apparently without loss of power. Tactile sensation was normal everywhere, but the point of a pin was miscalled at times in the left lower limb, as was also heat and cold stimulation in this limb. Vibratory sense and sense of position were normal.

Course of Disease.—The man returned Feb. 2, 1920. His condition had changed greatly. He had marked increase of power in his right lower limb and could move the limb more freely. He had no disturbance of sensation. Ankle clonus and the Babinski sign were still present on the right side. The right

thigh measured 38 cm., the left 41 cm.; the right leg measured 31 cm., the left 32 cm. The right lower limb was slightly rigid. The Wassermann test was reported delayed negative, less than ¼ inhibition.

On February 9 he had improved still more and had a great deal more power in his right lower limb, although he said both lower limbs seemed a little weak. There was now no decided loss of power in either lower limb.

The case evidently was one of organic change in the right side of the spinal cord, as shown by the ankle clonus and Babinski reflex. He probably had syphilis and syphilitic myelitis, and as a result of overwork the diseased blood vessels of the right side of the cord became congested. When he stopped working he allowed the circulation in the spinal cord to be restored in a considerable degree, but he was still weak in both lower limbs. The right crossed pyramidal tract and the right anterior horn of the lumbar region probably were affected.

Case 7.—*History.*—S. M., aged 33, came to the hospital Feb. 11, 1920. He had paralysis of the right lower limb which came on gradually two years before and which steadily increased. He had not had any pain. The left lower limb showed no loss of power. He sometimes had difficulty in voiding urine, and for the past year had had sexual impotence. He did not have headache, but he thought his eyesight was failing. He had three children, and his wife had had no miscarriages. So far as he knew, his symptoms had not changed during the past year.

Examination.—He had no Romberg sign. His gait was extremely spastic in the right lower limb, and he walked like a hemiplegic patient, swinging the limb from the hip. The pupils were equal and the reaction was good to light and in convergence. There were no symptoms in his face and upper limbs. The right upper limb was as normal as the left. The right lower limb was very weak and spastic, and when the limb was allowed to fall to the floor from the knee of the examiner it fell with the leg extended on the thigh and as a dead member, whereas the left lower limb fell in a normal manner. The left lower limb may have been a little weak, but not much so if at all. He used the left lower limb in a normal manner. The patellar and Achilles' reflexes were nearly equally exaggerated, but ankle clonus was present on the right and not on the left side. There was no disturbance of touch, pain, heat and cold sensations anywhere. Some of the Wassermann tests of the blood had been positive, and he had had twenty-four intravenous injections, but without improvement.

The right leg 8 inches below the top of the patella measured 13½ inches, and the left 14½ inches. The right thigh measured 3 inches above the top of the patella, was 15¾ inches, the left 17 inches; the right thigh 7 inches above the top of the patella measured 19 inches, and the left 19 inches.

This case seems to be similar to case 6 except that the monoplegia of the right lower limb was of long duration. The paralysis and spasticity of the right lower limb were intense. Babinski's sign was typical on each side. Persistent patellar clonus and persistent ankle clonus were present on the right side but not on the left side; the left patellar reflex was somewhat exaggerated. The left Achilles' reflex was about normal.

The pathologic condition was probably a syphilitic lesion of the spinal cord and did not appear to be of cerebral origin.

HISTOPATHOLOGY OF BRAIN ABSCESS

WITH REMARKS ON INTRASPINAL THERAPY *

GEORGE B. HASSIN, M.D.
Attending Neurologist, Cook County Hospital
CHICAGO

The pathologic changes in brain abscess are, as a rule, so manifest, even on macroscopic examination, that a correct diagnosis offers practically no difficulty, however insignificant the abscess may be. Probably this is the main reason for the scarcity of literature on microscopic brain changes resulting from abscess. Yet studies of the histologic changes are of great scientific interest, for they may clear up some as yet unsettled questions which are not only of academic, but also of practical, value.

Brain abscess perhaps brings to mind a circumscribed lesion more or less walled off by a membrane. Since time is required for a membrane to form, the walling off of an abscess necessarily must be a more or less gradual, subacute or chronic process that ultimately leads to transformation of a portion of brain tissue into a cavity filled with pus. This gradual replacing of the brain tissue by a walled off, encapsulated cavity is lacking in acute suppurative brain lesions. In these the suppurative process is rather diffuse, producing pronounced and extensive changes in the ganglion cells, glia tissue, nerve fibers and even in the vessels themselves.

MICROSCOPIC STUDY OF LESIONS OF BRAIN ABSCESS

Photomicrograph 1 (Fig. 1) gives some idea of widespread destruction. It represents a section of the brain cortex from a patient with a septic purulent meningitis secondary to an acute otitic abscess which ruptured into the brain, causing the patient's death on the twelfth day. The structure of the brain is totally changed. Instead of the regular layers of ganglion cells, there is a mass of strange elements which under low power can hardly be defined. Only blood vessels with somewhat thickened infiltrated walls and narrowed, thrombosed lumen, scattered fibrin fibers, erythrocytes and pus cells can be made out. Under a high power, there can be discerned fragments of somewhat changed nerve fibers, glia cells in a condition of ameboid degeneration and scattered hemorrhagic foci. Areas not directly affected by the pus invasion are much better preserved, though microscopically quite changed. In

* From the pathological laboratories of Cook County and Psychopathic Hospitals, Chicago.

these areas the blood-vessels exhibit dilated perivascular spaces infiltrated with lymphocytes and plasma cells, while the ganglion cells are either in a state of mild chromatolysis or so-called cloudy swelling, or altogether normal. In short, such spaces exhibit parenchymatous and interstitial changes typical of acute nonsuppurative encephalitis. These are the reactions of brain tissue in diffuse suppuration which has not had sufficient time to become encapsulated and which leads to total destruction of some elements (ganglion cells), partial destruction of

Fig. 1.—Brain section from a case of acute otitic abscess that ruptured into the brain killing the patient on the twelfth day of his illness. The black spots are vessels, thrombosed and with thickened walls, surrounded in some places by fibrin fibers. The numerous dots are glia nuclei. (Frozen section. Alzheimer-Mann stain, × 75.)

others (nerve fibers, glia cells) and progressive vascular changes (hyperemia, thickening and infiltration of the vascular walls, new formation of capillaries, etc.).

Source of Connective Tissue in Brain Tissue.—Perhaps of greater interest are the findings in cases of encapsulated abscess. The capsule of an old abscess may be incredibly thick. For instance, in one of Bassoe's cases of eight years' duration, demonstrated before the Chi-

cago Neurological Society, the thickness was one quarter of an inch, very dense and fibrous, much resembling the dura. In fact, the first impression gained from a superficial examination was that the membrane was an outgrowth from the dura. Thus we are confronted by a peculiar phenomenon: growth of a large mass of hard connective tissue in brain tissue, where under normal conditions, connective tissue is solely confined to vessels and the tender prolongations of the pia. Naturally, it is desirable to determine how and from where such an

Fig. 2.—A surface (longitudinal) section of the inner layer of the capsule, eight years old. The connective tissue fibers crossing and intercrossing each other form dense meshes with narrow interspaces enclosing a few cellular elements; there is a total lack of blood vessels. The entire structure very much resembles a fibrous scar tissue. (Combined Bielschowsky-Alzheimer-Mann stain. Paraffin section, × 45.)

abnormal, unusual and excessive amount of connective tissue growth develops. This problem I have tried to solve.[1] The study of my material (young membranes) apparently demonstrated that the growth of connective tissue arose exclusively from the blood elements, the lymphocytes, which by gradual and progressive transformation turned

1. Hassin, G. B.: Histopathologic Studies on Brain Abscesses, Med. Rec. **93**:91 (Jan. 19) 1918.

into fibrobiasts, and then into connective tissue fibers, finally to become a solid membrane.

Through the kindness of Dr. Peter Bassoe, I have had the opportunity of studying much older abscess membranes. One, about eight years old, enveloped a large abscess that resulted from a bullet lodged in the brain. In three other cases the membranes were comparatively younger, but their macroscopic and microscopic aspects were much alike. The membranes, as well as various portions of the brain and

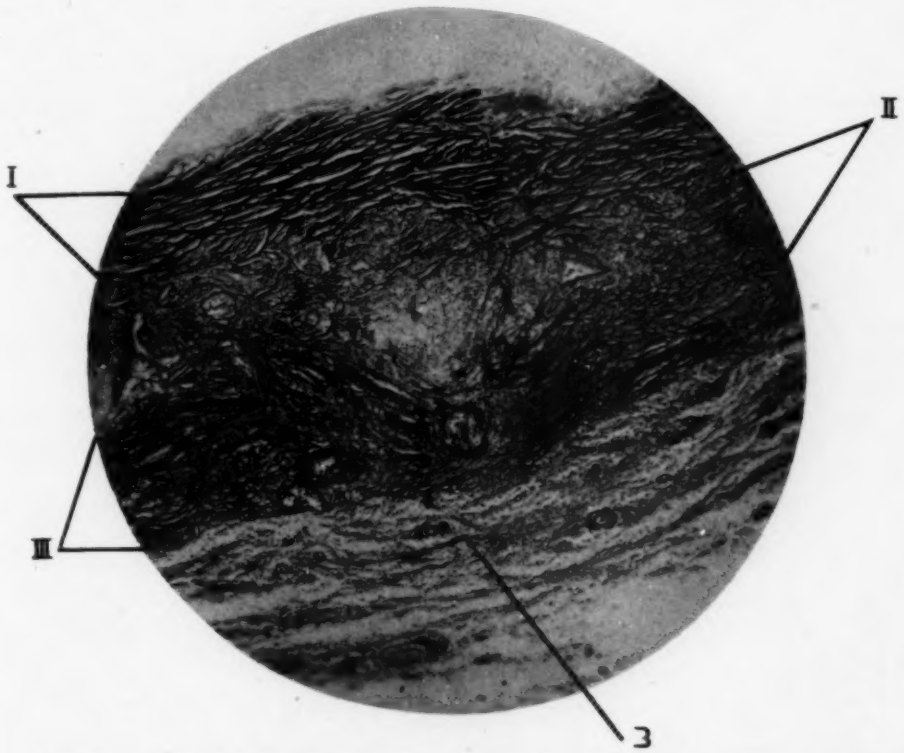

Fig. 3.—Transverse section of the eight year old capsule. The external layer (III) consists, like the inner one, of connective tissue but shows a number of vessels that are absent in the internal layer (I). II—the middle layer also reproduced on a surface section on the photomicrograph. IV—B, brain tissue shows numerous small vessels infiltrated with fat. (Stain same as in Figure 2, × 45.)

cerebellum, have been studied on celloidin, paraffin, and frozen sections with various staining methods. The capsules were examined in transverse and longitudinal or surface sections, the respective layers having been taken up separately. The inner layer (Fig. 2) directly surrounding the abscess presents a mass of collagen, powerful fibers crossing and intercrossing, forming an irregular network containing

numerous interspaces. The latter usually enclose a few cellular elements, or rather remnants of various cells which cannot be well defined. They mostly appear shrunken, some being cystic and vacuolated, some oblong and flattened, with a very pale, hardly visible nucleus. Few of them show the presence of fat. Blood vessels in this layer are lacking, as well as polymorphonuclear cells, pus cells, fibroblasts or other inflammatory elements. Somewhat similar in structure is the external layer (Fig. 3). It also contains a wealth of collagen fibers, which, though

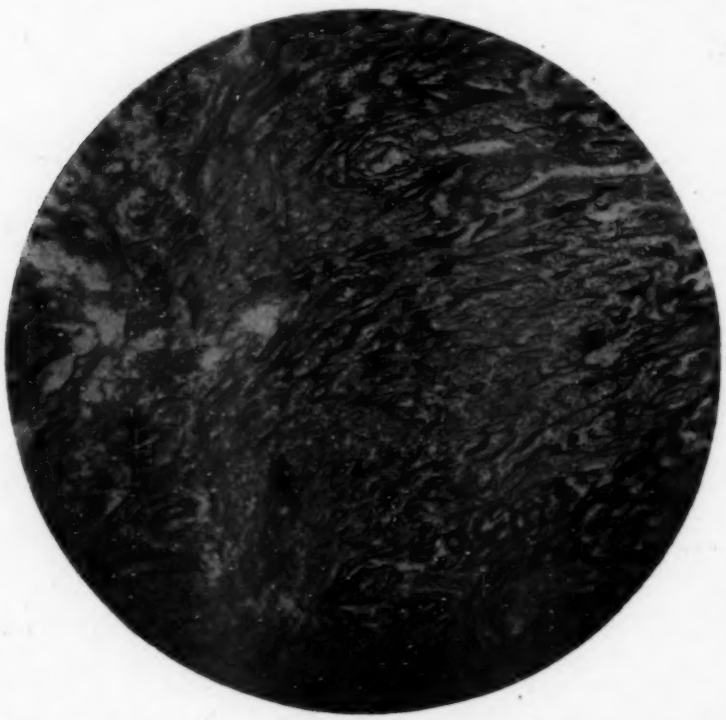

Fig. 4.—Surface section of the middle layer of the eight year old capsule. The predominance of vessels and cellular elements (in the pale areas) is quite marked. (Stain and magnification the same as in Figures 2 and 3.)

somewhat less thick, equally form dense meshes packed with an enormous amount of cellular elements (plasma cells, gitter cells, fibroblasts and some lymphocytes). Vessels here are more numerous, their adventitial spaces are dilated, but blood cells, pus elements, polymorphonuclear cells and signs of inflammation are totally lacking. Between these two membranes there can be discerned (Figs. 3 and 4) the third layer which shows a wealth of cellular elements and vessels. This membrane also contains connective tissue fibers, but these are much more tender and delicate than those of the external and internal layers.

In some places it consists solely of a great mass of fat granule bodies which, with Herxheimer scarlet red stain, appear (Fig. 5) as black masses. Being comparatively poorer in collagen fibers and richer in blood vessels and cellular elements (plasma cells, fibroblasts), this membrane is evidently less organized, and therefore less powerful than the outer and inner layers. The difference of structure in the three layers making up the abscess capsule can be seen from a glance at Figure 3 which represents a transverse section of the capsule. The

Fig. 5.—Surface frozen section of the middle layer of Figure 3 shows vast accumulations of fat as black masses. (Herxheimer scarlet red stain, × 100.)

inner and outer layers, similar in structure, are separated by a paler zone rich in gitter cells and blood vessels. The first two appear like a scar, while in the middle the scar formation is hardly advanced. The cicatrization apparently is obtained in those zones where it is most needed: first, in the surface next to the abscess to hold it in check and, second, on the border of the brain substance to prevent its contamination, or invasion by pus. These three layers in a capsule can be still more easily distinguished in a case of but a few months' duration. The membrane in this case (Fig. 6) also shows a powerful inner layer,

a less powerful, though fully developed external layer and a middle layer rich in vessels and cellular elements.

Comparing Figures 3 and 6 one can easily see the difference between these two capsules: the former (Fig. 3) is almost totally organized, while the latter is but partially so. Had the patient in the latter case lived longer, the capsule would undoubtedly have assumed the features

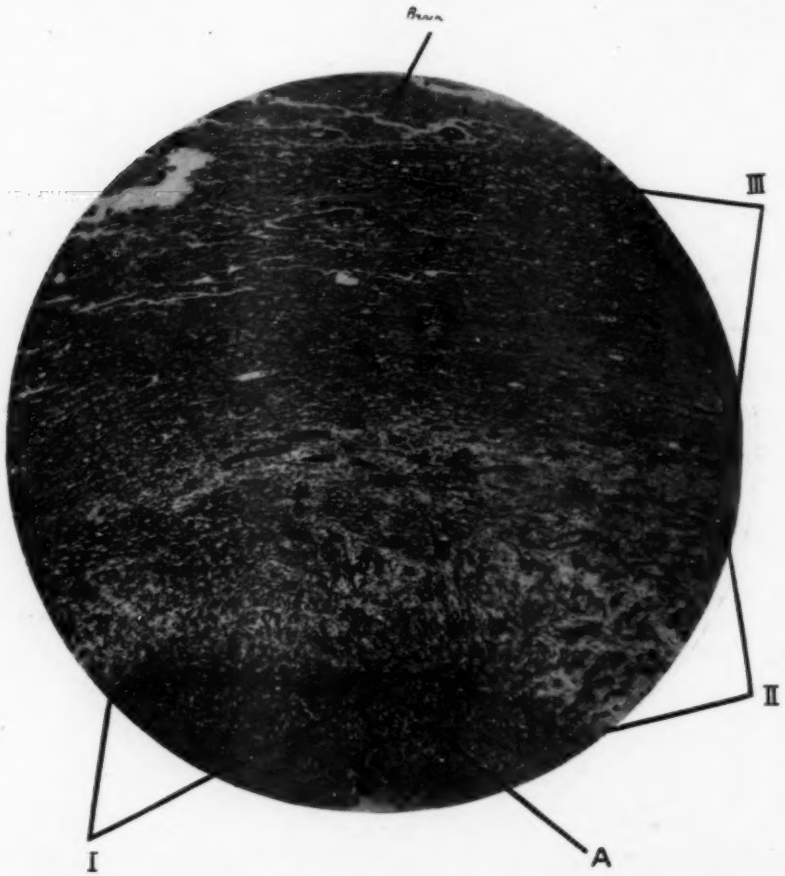

Fig. 6.—Transverse section of the younger capsule (several months old) I, II, III, respective layers; A, the abscess. The top of the picture represents a practically normal brain tissue with several infiltrated vessels. Like the much older capsule reproduced in Figure 2, it shows the identical organization of the inner (I) and external (III) layers; while the middle (II) layer, rich in vessels, is less organized. (Van Giesen stain, × 30.)

of the one in the former case. Whether young or old, once organized, the capsule surrounding the abscess forms an excellent protection for the brain tissue, which is wholly preserved (Figs. 6, 7 and 8). It exhibits no pathologic changes in the ganglions, glia cells or nerve

fibers, but the vessels and the pia-arachnoid are decidedly altered. While the lumen of a vessel, its intima and the middle layers are normal, the adventitia with its space (Virchow-Robin) is enormously developed (Fig. 9) and made up of numerous bands of connective tissue fibers with narrow interspaces packed with plasma cells. The latter can be easily recognized in the photomicrograph (Fig. 9), espe-

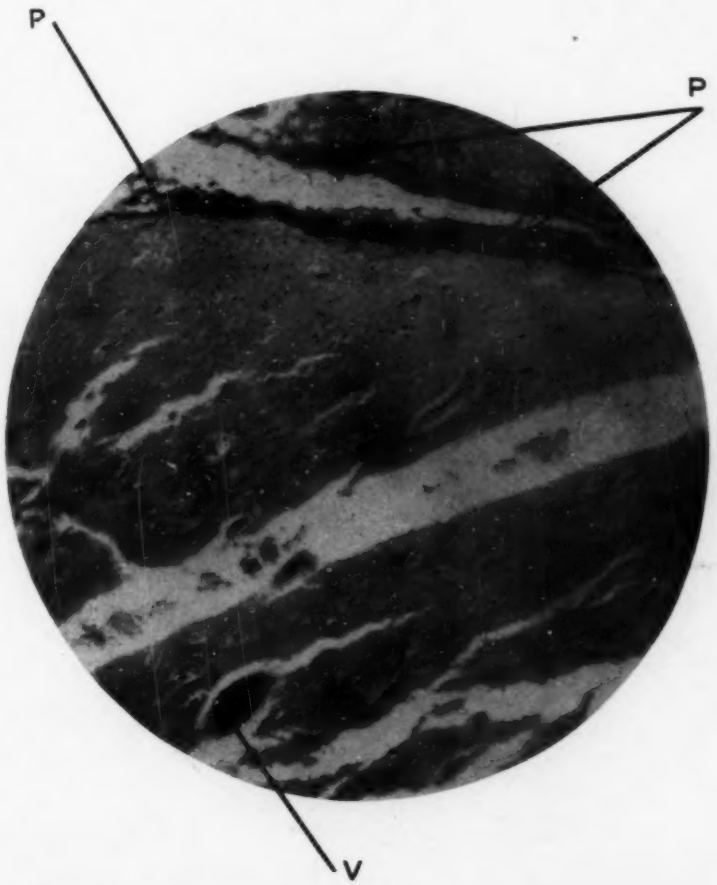

Fig. 7.—Between the much infiltrated blood vessel (V) and the pia (P) (especially its lower branch) is situated an apparently normal brain tissue. The infiltration elements are mostly fat granule bodies and other elements reproduced in Figures 8 and 10. (Bielchowsky-Alzheimer-Mann stain, × 100.)

cially with the help of a hand lens. Aside from plasma cells and their modifications, young fibroblasts, there are also present gitter cells filled with fat globules. Numerous in the external portions of the adventitia, the cellular elements are scarce in the inner portions, where they are replaced by collagenous fibers, which totally fill up the distended adventitial spaces (Fig. 9). In short, the adventitia is in a hyperplastic state

surrounding the vessels like a muff and invariably containing fat. It represents an extinct inflammatory condition which ultimately resulted in the formation of a fibrous ring around the vessel.

Far removed from such a vessel (Figs. 7 and 8), and separated from it by normal brain tissue, is the pia arachnoid. At the first glance this appears to be infiltrated in some places. However, closer examination (Fig. 8) shows that the infiltration did not involve the

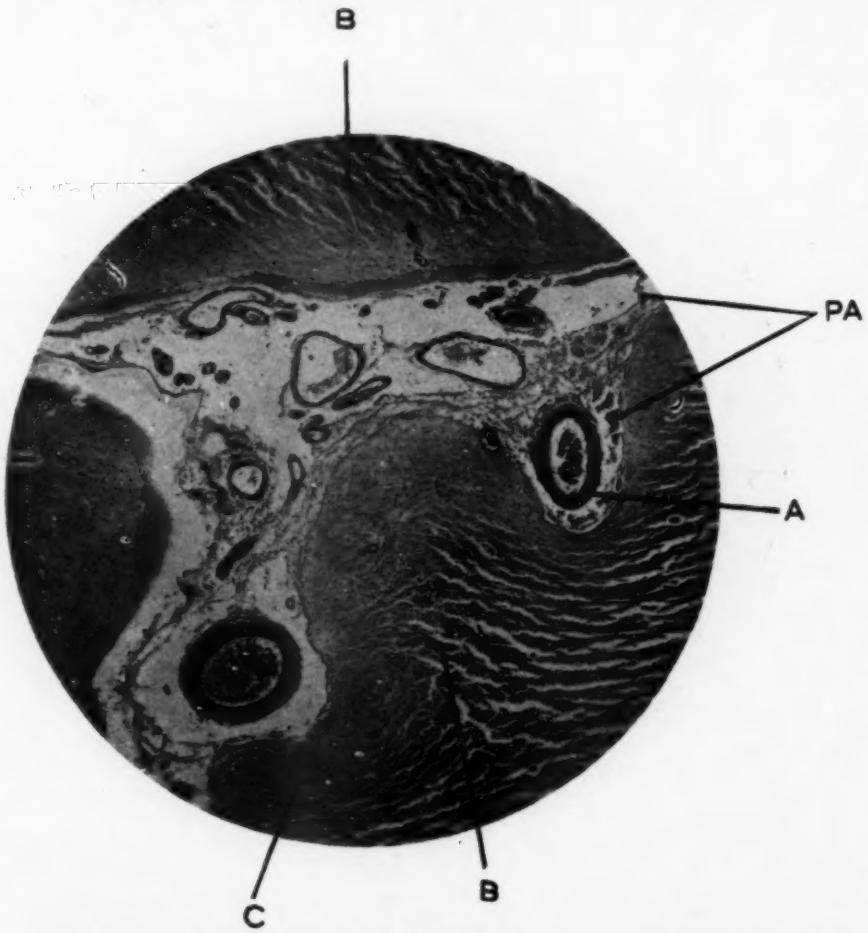

Fig. 8.—P A, pia arachnoid, showing the dilated and infiltrated subarachnoid space as well as numerous vessels; A, artery with an infiltrated adventitial space; B, brain tissue without any changes, except minute capillaries slightly infiltrated with fat. C, infiltrated capillary. Use hand lens. (Same stain as in Figure 7, ×33.)

pia at all, this membrane being normal. The infiltration is rather confined to the distended subarachnoid space as well as to the numerous vessels. The infiltration elements are partly lymphocytes, but mostly macrophages and gitter cells (Fig. 10). The macrophages and gitter

cells are very large, the former enclosing various débris of lymphocytes, nuclei, etc., while the gitter cells are packed with lipoid, or fat-like substances. Evident signs of inflammation, hemorrhages, fibroblasts, plasma cells, etc., are lacking in the pia-arachnoid. The lipoid substances, as well as cell remnants, enclosed within the macrophages thus can be located in the subarachnoid spaces even when they are widely separated from the abscess and its capsule. The probability is strong that these lipoid substances came into the subarachnoid space from the capsule, its vessels and their perivascular spaces. In other words, it is most probable that the fat found in such abundance in the

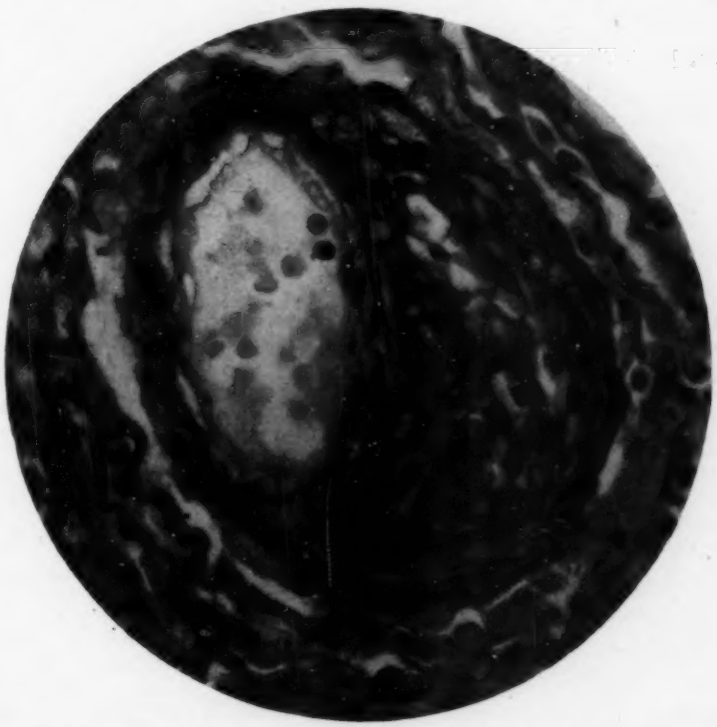

Fig. 9.—Transverse section of a blood vessel (vein) from the vicinity of a capsule of an eight year old brain abscess. The patent lumen shows red cells and two lymphocytes; the adventitial, greatly dilated, space exhibits a vast amount of connective tissue fibers resembling a scar and containing in the interspaces numerous plasma cells, especially in the external areas. Many plasma cells are assuming the shape of fibroblasts. Use a hand lens. (Same stain as in Figure 7, × 600.)

capsule reached the subarachnoid space by way of the perivascular spaces or—which is the same thing—that the contents of the perivascular spaces were emptied and discharged into the subarachnoid cerebral space. In no other way could the presence in the latter of gitter cells, macrophages and lymphocytes be explained, as a normal pia-arachnoid is totally devoid of such elements.

DIRECTION OF FLOW OF BRAIN TISSUE FLUIDS

But to enable pathologic elements to reach the subarachnoid space from the depths of the brain tissue, there must be some pathway leading from the latter to the former; this pathway is composed of the perivascular spaces of Virchow-Robin. That they form such a connecting link is generally admitted, and Weed,[2] by his remarkable experimental work, conclusively proved that the flow of the perivas-

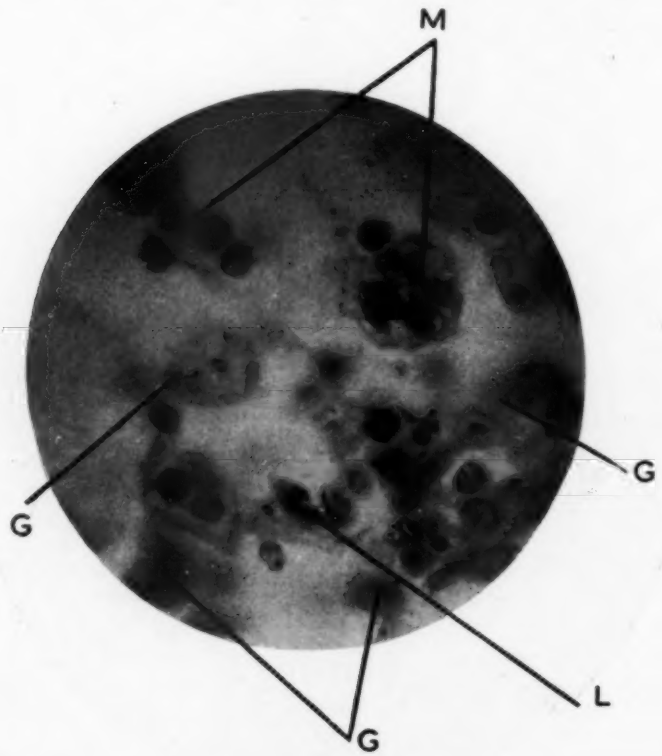

Fig. 10.—Types of infiltration cells in the subarachnoid space reproduced in Figure 8. M, macrophages; G G, Gitter cells; L, lymphocytes. (Same stain as in Figure 7, × 1200.)

cular contents is from the brain to the subarachnoid space. For his experiments Weed used 1 per cent. solution of equal amounts of iron

2. Weed, L. H.: Studies on Cerebrospinal Fluid. II. The Theories of Drainage of Cerebrospinal Fluid with an Analysis of the Methods of Investigation, J. Med. Res. **26**:21, 1914-1915. III. The Pathways of Escape from the Subarachnoid Spaces with Particular Reference to the Arachnoid Villi, Ibid. **26**:51, 1914-1915; IV. The Dual Source of Cerebrospinal Fluid, Ibid. **26**:93, 1914-1915; An Anatomical Consideration on Cerebrospinal Fluid, Anat. Rec. **12**:461 (May) 1917-1918.

ammonium citrate and potassium ferrocyanid. This fluid, isotonic with the body fluids, was injected under long continued pressure of from 130 to 180 mm. of water, into the lower thoracic and upper lumbar regions, or into the arachnoid cistern about the cerebellum of cats and dogs. After the injection was completed the head of the animal was severed from the body and placed in a 40 per cent. solution of formaldehyd to which a from 1 to 5 per cent. solution of hydrochloric acid was added. The resulting precipitate of ferric ferrocyanid in the form of bluish granules (Prussian blue) could easily be identified wherever the fluid had been absorbed. The course of the absorption of the injected solution can be assumed, as Weed rightly concluded, to be the true pathway normally pursued in the absorption of the cerebrospinal fluid, as the results are obtained with nontoxic solutions practically isotonic with the body fluids. These pathways, as indicated by the deposits of the Prussian blue granules, prove to be the pial meshes, the subarachnoid space, the dural spaces, the pacchionian bodies and the sinuses (longitudinal, cavernous) as well as the perineural spaces of some cerebral nerves, diploe vessels and lymph channels (outside of the cranium, but none in the brain tissue proper). Practically the same pathways could be traced in a case of metastatic carcinoma of the cerebral meninges[3] in which, as in Weed's experiments, their invasion by carcinomatous cells was not accompanied by that of the brain. Only by using high pressure in his injections, did Weed succeed in finding the Prussian blue granules in the adventitial spaces of the brain vessels.

These experimental and pathologic facts apparently prove that the brain substance, under normal conditions, does not obtain anything from the subarachnoid space whatever its content may be—Prussian blue, cancer cells or a solution of arsphenamin. This contention is supported by Figure 11, a photomicrograph from a case of cerebrospinal meningitis. The pia is greatly infiltrated by plasma cells, the subarachnoid space is distended and separated from the arachnoid by a mass of pus cells, while the cerebellar tissue does not show a single pus cell, plasma cell or any other kind of abnormality. In some way the plasma and pia cells are kept, as it were, from penetrating into the cerebellar substance. This would not be the case had the flow been from the meningeal spaces toward the cerebral or cerebellar substance.

While this finding, together with the above mentioned results of Weed's work, seem to prove that the cerebrospinal fluid or the content of the subarachnoid space does not flow toward the brain, there are comparatively few and less convincing facts tending to show that the

3. Hassin, G. B.: Histopathology of Carcinoma of the Cerebral Meninges, Arch. Neurol. & Psychiat. **1**:705 (June) 1919.

current of the brain tissue fluids is toward the subarachnoid space and away from the brain. These brain tissue fluids are normally present in the adventitial or perivascular spaces of Virchow-Robin, which spaces it is generally admitted, are connected with the subarachnoid space, the container of the cerebrospinal fluid. Spina's,[4] Foerster's [5] and Weed's [2] experiment's brought out some striking evidence in favor

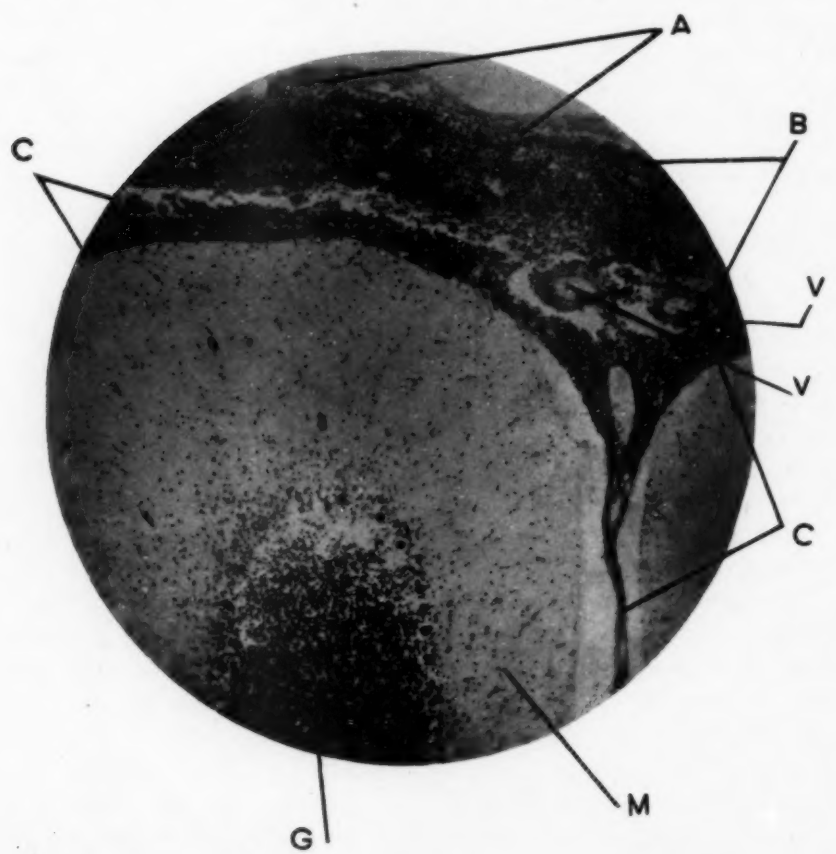

Fig. 11.—Pia arachnoid and subarachnoid space from a case of cerebrospinal meningitis. A, arachnoid; B, subarachnoid space packed with pus cells; C, pia infiltrated with plasma and mesothelial cells; V V, blood vessels of the pia with an infiltrated adventitia; M, molecular layer of the cerebellum, free from any foreign elements; G, granular layer of cerebellum surrounded by Purkinje cells. The molecular layer (M) shows two prominent capillaries with densely stained endothelial cells. (Toluidin blue stain, × 60.)

4. Spina, A.: Experimentelle Untersuchungen über die Bildung des Liquor Cerebrospinalis, Arch. f. d. ges. Physiol. **76**:204, 1899.

5. Foerster, E.: Experimentelle Beiträge zur Lehre der Phagocytose der Hirnrindenelemente, Nissl's Arbeiten **11**:173, 1908.

of the opinion that the current is from the adventitial spaces toward the subarachnoid space. Spina found beads of fluid on the surface of the cerebrum after he had produced increase of pressure in the brain. Foerster found india ink granules in the subarachnoid space after injecting sterile india ink solution into the cerebral substance, and Weed, as already noted, found granules in the brain vessels when he made injections under high pressure. The pathologic findings in brain abscess conclusively show, in my opinion, the correctness of the view of the foregoing investigators that the flow is from the adventitial spaces of the brain toward the subarachnoid space. The enormous masses of fat in the capsule, as shown in Figure 5, are discharged or transported to the subarachnoid space by the way of the perivascular spaces of the cerebral vessels. The fat substance in the subarachnoid space could not reach it in any other way. Neither could the presence of fat in the perivascular spaces of the cerebral blood vessels themselves be explained, unless it came from its source of supply — the abscess and its capsule. This outward flow of brain tissue fluid seems to me to be but a natural and unavoidable process. The waste of the activity of the brain elements must be removed in some manner, and as the central nervous system lacks lymphatics, this removal is accomplished by the system of closed formations, known as Virchow-Robin's perivascular spaces. It follows that any substance injected into the brain substance will be eliminated into the subarachnoid space probably without being assimilated by the ganglion cells. On the other hand, any appropriate substance injected into the subarachnoid space will be discharged, as Key and Retzius and Weed showed, through the arachnoid villi (pacchionian bodies) into the blood stream without reaching the brain substance at all.

ABSENCE OF PHYSICLOGIC SUPPORT FOR INTRASPINAL THERAPY

The practical conclusion is obvious; namely, that intraspinal therapy of cases in which especially the parenchyma of the brain is deeply involved, as in general paresis, has no physiologic support. In those cases in which the trouble is in the subarachnoid space itself, as in cerebrospinal meningitis, the injection is wholly justified, for given in suitable doses it may produce results. But as used in intraspinal therapy, in very small doses (fractions of a grain of arsphenamin), therapeutic results can hardly be expected. I believe that the favorable reports of the Swift-Ellis method of treatment by some reliable observers can be explained, not by the action of the intraspinal injections, but by that of the intravenous ones. Arsenic injected into the blood may reach the spinal fluid and the meninges, as demonstrated

among others by G. W. Hall,[6] while Tilney[7] found the membranes stained with trypan blue after injecting it intravenously. The foregoing observations show that a drug injected into the veins may reach the meninges, and there is no object in trying to accomplish the same result by the intraspinal route.

One more point deserves attention and at least a brief discussion. It has been almost generally admitted, especially by French and English observers (Laignel-Lavastine, Carrier, Pierret, Wilkes and McEwen) that every meningitis is an encephalitis, that every inflammation of the pia is accompanied by inflammation of the brain tissue proper. This opinion has been shown to be erroneous by the histologic research work of Ranke,[8] while the foregoing considerations relative to the connections of the meningeal spaces with those of the brain, as well as to the flow of the brain tissue fluids, decidedly favor Ranke's views. Cases of pure leptomeningitis are not accompanied by encephalitis. The superficial layers of the cortex may be invaded from the meninges by pathologic elements and exhibit some inflammatory phenomena, but the latter will spare the deeper strata of the brain as well as its smallest blood vessels. If the latter are also involved, we have an encephalitis which is independently caused like the meningitis by the same morbid agent. These considerations also apply to poliomyelitis in which the parenchymatous and interstitial changes in the spinal cord are associated with meningitis. To consider the former as a product of the latter, that is, to assert with Harbits and Scheel,[9] for instance, that the pathologic changes of the spinal cord in poliomyelitis are caused by the leptomeningitis, is not in accord with the facts, for both the meningeal and spinal cord changes are due to one cause. Such, in brief, are some of the facts that can be more or less definitely established from histopathologic studies of suppurative conditions of the brain.

31 North State Street.

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6. Hall, G. W.: Report before the Chicago Neurological Society, Feb. 19, 1920.

7. Tilney, Fred: Long Island M. J. **8**:121, 1914; J. Nerv. & Ment. Dis. **41**:454, 1914.

8. Ranke, Otto: Beiträge zu der Lehre von Meningitis Tuberculosa, Nissl's Arbeiten **2**:334, 1908.

9. Harbitz, F., and Scheel, O.: Pathologisch-Anatomische Untersuchungen über akute Poliomyelitis und verwandte Krankheiten, Christiania, 1907.

FURTHER OBSERVATIONS ON THE PRESENCE OF ARSENIC IN THE SPINAL FLUID

GEORGE W. HALL, M.D., RUSSELL J. CALLENDER, M.D.
AND
EDWARD C. HOLMBLAD, M.D.
CHICAGO

In 1915 one of us reported some work on this subject (G. W. H.),[1] and in 1917 Hall, Schlegel and McNally made further examinations (Marsh's test) with the following results (unpublished): In thirteen cases the spinal fluid was withdrawn twelve hours after the intravenous administration of either novarsenobenzol or neodiarsenol brand of arsphenamin in 0.6 gm. doses and in four instances the presence of arsenic was noted. In twelve cases treated similarly and the spinal fluid withdrawn twenty-four hours after the intravenous medication, no arsenic was found.

March 15, 1919, we reported to the Chicago Neurological Society a new procedure which we hoped would facilitate the passage of arsenic into the spinal fluid after its intravenous administration. We now report in full the results of this work and the method used.

PROCEDURE AND RESULTS OF ARSENICAL TESTS OF SPINAL FLUID

We chose patients who, so far as we know, had never been subjected to any form of intravenous medication except as noted in the accompanying table. Serum from the patient's blood was injected into the spinal canal and at intervals of from four to eleven hours afterward, an arsenical preparation was given intravenously. At different intervals after the intravenous injection, the spinal fluid was withdrawn and tested for arsenic. We hoped to develop a safer and more effective method than that of Swift-Ellis. The table gives our results in eighteen cases.

Mehrtens and MacArthur, who used a similar technic, report the presence of arsenic in several instances in quantities less than the practical delicacy of the test will permit. According to their table, arsenic was detected in quantities smaller than 1 micromilligram.[2]

1. Hall, George W.: The Presence of Arsenic in the Spinal Fluid, J. A. M. A. **64**:1384 (April 24) 1915.

2. Mehrtens, H. G., and MacArthur, C. G.: Therapy of Neurosyphilis Judged by Arsenic Penetration of Meninges, Arch. Neurol. & Psychiat. **2**:369 1919.

FINDINGS IN TESTS FOR PRESENCE OF ARSENIC IN THE SPINAL FLUID IN EIGHTEEN CASES

Clinical Diagnosis	Previous Arsenical Medication	Blood Wassermann Test	Spinal Fluid		Autoserum Intraspinously				Arsphenamin Injection Intravenously		First Withdrawal of Spinal Fluid				Second Withdrawal of Spinal Fluid				Third Withdrawal of Spinal Fluid			
			Wassermann Test	Globulin Test Cell Count	Time Given	Amount in C.c.	Cell Count	Control Arsenic Test	Hours After Serum Injection	Amount	Hours After Arsphenamin Injection	Amount in C.c.	Cell Count	Arsenic Test	Hours After Arsphenamin Injection	Amount in C.c.	Cell Count	Arsenic Test	Hours After Arsphenamin Injection	Amount in C.c.	Cell Count	Arsenic Test
4/11/19 Early syphilis	None	4+	0	Not recorded 4/12/19	4/22/19 12 noon	10	360	Negative	5¼	0.6 Neodiarsenol	7½	10	4,000	Positive, trace	18½	8	2,000	Negative	40¼	8	360	Negative
4/18/19 Tabes dorsalis	None	0	4+	+ 154 4/23/19	5/5/19 5 p.m.	12	110	Negative	4½	0.6 Neodiarsenol	12¾	10	1,115	Negative	22	10	280	Negative	38	10	210	Negative
5/2/19 Tabes dorsalis	None	0	0	Not recorded	5/15/19 12 noon	12	20	Negative	5	0.6 Neodiarsenol	6	10	230	Negative	41	10	74	Negative	113	10	35	Negative
5/2/19. Tabes dorsalis crisis	None	0	4+	+ 56 5/2/19	5/5/19 5:30 p.m.	15	30	Negative	5	0.6 Neodiarsenol	3½	10	500	Negative	9½	10	1,260	Negative	25½	10	480	Negative
6/2/19, Cerebrospinal syphilis	None	0	4+	++ 124 6/2/19	6/4/19 2:15 p.m.	12	88	Negative	3¾	0.6 Neodiarsenol	5½	8	1,110	Positive	17½	10	180	Positive	39½	10	45	Positive
4/21/19 Tabes dorsalis	++ Intravenous, Intraspinal 4/1/17	0	4+	++ 165 5/9/19	6/4/19 2:30 p.m.	12	02	Negative	4¼	0.6 Neodiarsenol	5¾	5	700	Positive	17¾	8	1,070	Negative	39¼	10	80	Negative
5/9/19 Tabes dorsalis	None	0	0	++ 35 5/9/19	6/4/19 2 p.m.	15	16	Negative	5¼	0.6 Neodiarsenol	5	8	1.800	Negative	17	10	2,880	Negative	39¼	10	420	Negative

5/17/19 Cerebrospinal syphilis	None	0	4+	Not recorded 5/19/19	6/4/19 1:45 p.m.	16	1,648	Negative	4¾	0.6 Neodiarsenol	5¾	8	2,070	Negative	17¾	10	2,140	Negative	40	15	2,000	Negative
6/6/19 General paresis	None	4+	4+	+ 78 6/7/19	6/10/19 4:30 p.m.	10	90	Negative	10¾	0.6 Metz Neosalvarsan	8	10	400	Negative	17½	8	1,830	Negative	35¼	10	440	Negative
5/20/19 Taboparesis	++ Intravenous, Intraspinal 6/1/18	0	4+	++ 14 5/20/19	6/10/19 4:15 p.m.	15	35	Negative	11¼	0.6 Metz Neosalvarsan	8	10	400	Negative	17¼	10	1,320	Positive	35¼	10	860	Positive
5/12/19 Cerebrospinal syphilis	None	0	0	++ 100 5/13/19	6/10/19 4:45 p.m.	10	450	Negative	11¼	0.6 Metz Neosalvarsan	8	10	1,180	Negative	17¾	10	1,240	Negative	35¼	10	180	Negative
5/26/19 General paresis	None	0	4+	+ 240 5/26/19	6/10/19 5:15 p.m.	12	280	Negative	12	0.6 Metz Neosalvarsan	7¾	10	220	Negative	17½	15	Too bloody	Negative	35¼	10	Too bloody	Negative
Tabes	None	...	...	20	10/19/19 10:30 a.m.	15	.....	Negative	6	0.6 Neosalvarsan	1	10	470	Negative	7	10	480	Negative	24	10	210	Negative
Tabes	None	...	...	11	10/23/19 9 a.m.	15	.....	Negative	6	0.6 Neosalvarsan	1	8	4,000	Negative	6	8	3,710	Negative	18	15	980	Negative
Cerebrospinal syphilis	None	...	...	66	11/6/19 9 a.m.	10	.....	Negative	6	0.6 Neosalvarsan	1	8	322	Negative	7	8	134	Negative	18	5	100	Negative
Tabes	None	...	...	6	10/16/19 11:30 a.m.	10	.....	Negative	5½	0.6 Neosalvarsan	1½	8	75	Negative	6	8	30	Negative	24	10	44	Negative
Tabes	None	...	...	10	10/16/19 10:30 a.m.	15	.....	Negative	5	0.6 Neosalvarsan	1¼	10	33	Negative	6	8	150	Negative	23½	10	200	Negative
Cerebrospinal syphilis	None	...	...	360	10/16/19 11 a.m.	8	.....	Negative	6½	0.6 Neosalvarsan	..	..	.....	.....	..	..	.....	.....	23½	10	.....	Negative

Mr. R. Bodmer, in his discussion of John Webster's paper, on the "Excretion and secretion of Salvarsan and Neosalvarsan," stated that he had had a good deal of experience in the estimation of small quantities of arsenic in toxicologic cases, but he said that the accurate estimation of such small quantities as 0.01 mg. in 100 gm. seemed to him a rather difficult matter. Mr. Chapman remarked, in discussing the same paper, that 0.002 mg. was about the safe limit with zinc and hydrochloric acid.[3]

In our investigation, arsenic was determined in the spinal fluid by the Gutzeit method which is described in the United States Pharmacopeia and in the Journal of the Association of Official Agricultural Chemists.[4] In this method the detection of arsenic depends on the reaction of arsin with the bromid or chlorid of mercury whereby a color is produced. The color ranges from lemon yellow through orange yellow to reddish brown. Preliminary treatment of the substance in question is necessary in order to change the arsenic into an inorganic salt from which it is readily converted into arsin by means of nascent hydrogen. The change to the inorganic salt may be accomplished by strong oxidizing reagents, such as nitric and sulphuric acids, hydrochloric acid and potassium chlorate, or by bromin water in the presence of hydrochloric acid. The use of bromin as an oxidizing agent for the digestion of organic material in the determination of arsenic is suggested by C. S. Smith, in the Bureau of Chemistry Circular No. 102, 1912. In the same circular is mentioned the superiority of mercuric bromid over mercuric chlorid for the preparation of the sensitized paper used to detect the presence of arsin.

All reagents must be free from arsenic. In each case a blank must be run to make sure of this fact. We have made no attempt to determine the amount of arsenic present, but simply its presence or absence. Approximately 10 c.c. of spinal fluid are digested in a 150 c.c. flask on a steam bath for two hours with 5 c.c. of concentrated hydrochloric acid. The mixture is allowed to cool and 5 c.c. of saturated bromin water are added. The flask is then replaced on the steam bath and allowed to remain until all traces of bromin have disappeared. After cooling, the solution is transferred to a bottle and diluted with water to make about 25 c.c. In the stopper of the bottle is fitted a small piece of glass tubing in which is placed a pledget of cotton saturated with 5 per cent. lead acetate solution. Into the end of the tube is fitted a smaller tube in which is placed a strip of filter paper 4 mm. wide, previously treated with a 5 per cent. alcoholic solution of mercuric

3. Analyst **41**:231, 1916.

4. United States Pharmacopeia, Philadelphia, P. Blakiston's Son & Co. **9**:584. J. Assn. Official Agric. Chemists **2**:171, 1916.

bromid. When all is ready, three drops of a 40 per cent. solution of stannous chlorid in concentrated hydrochloric acid and 5 c.c. of a 15 per cent. solution of potassium iodid are added to the contents of the bottle. Finally from 5 to 7 gm. of granulated zinc are added. As soon as the zinc is added, the stopper is quickly replaced and any change in the paper noted. The colors obtained are compared with the color obtained from a blank run at the same time and under the same conditions. Using the best reagents procurable, a slight yellow color often develops on the paper of the blank which in the hands of the inexperienced might be considered a trace of arsenic. However, if the experimental apparatus shows no deeper coloration than a blank run under the same conditions, no arsenic should be reported.

The absolute delicacy of this method is set at 0.00008 mg. or 0.08 micromilligrams of arsenic trioxid.[5] The practical delicacy with bands of paper 4 mm. wide is 0.001 mg. or 1 micromilligram, that is, one part per million on a gram sample. In analytic chemical work few methods have greater delicacy than this test. The usual clinical laboratory methods seldom if ever approach its accuracy.

If this method is to be used in clinical work more than the usual precautions must be used. Special care must be taken to prevent contamination with arsenic either in withdrawing the fluid or in making the test.[1] Otherwise, arsenic may be detected in the spinal fluid derived from other sources than the blood stream. For example, arsenic determinations made in a room in which arsphenamin mixtures are prepared for injections are liable to show the presence of arsenic. In our own work we have tried to avoid any possible source of contamination with arsenic.

CONCLUSIONS

Arsphenamin given intravenously in 0.6 gm. doses may be detected in the spinal fluid in from 25 per cent. to 35 per cent. of the cases.

Irritation of the meninges by the injection of autoserum into the spinal canal does not increase this percentage.*

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5. Autenrieth-Warren: Detection of Poisons, Ed. 4, Philadelphia, P. Blakiston's Son & Co., p. 235.

*The laboratory technic was carried out by Clayton S. Smith, Ph.D., Department Laboratory of Physiology and Pharmacology, Northwestern University Medical School.

A REPORT OF ELEVEN CASES OF CERVICAL SYMPATHETIC NERVE INJURY, CAUSING THE OCULOPUPILLARY SYNDROME

STANLEY COBB, M.D.
BOSTON
AND
HUNTER W. SCARLETT, M.D.
PHILADELPHIA

The report of the following series of cervical sympathetic nerve lesions, due to gunshot wounds, observed in the U. S. Army General Hospital No. 11, was prompted by the comparatively large number coming under our care at one time, and by the relatively few cases noted in military literature to date. It may be that more such cases have not been recorded during the present war, first, because in the stress of work they were overlooked; secondly, because of their slightly defined symptoms; and lastly, because it is possible that no evidence of a Claude Bernard-Horner syndrome appeared until some weeks or months after injury, the patients by that time having passed from under medical supervision. Our patients, on the other hand, had in some instances been wounded from six to nine months before admission to the hospital, and sufficient time had elapsed for symptoms to develop. Then too, we had the opportunity to go over each case thoroughly, and to keep the patient under observation.

Heretofore the majority of sympathetic paralyses have occurred in civil life, and have been due to causes other than traumatic, such as cervical rib, aneurysm, enlarged glands, mediastinal tumors, and other conditions; although some have been due to injuries, and a few have been recorded in times of war. The earliest data we have on this subject, especially from the standpoint of ocular symptoms, dates back to 1727, when Pourfour du Petit [1] made his experiments on the cervical sympathetic nerve. In 1851, Budge [2] localized the center for the pupillodilator fibers between the sixth cervical and the fourth dorsal vertebrae. Claude Bernard,[3] in 1858, working on animals, observed the

1. Du Petit, Pourfour: Memoire sur la portion cervicale du nerf grand sympathique; Memoires de l'Academie des Sciences, 1727.

2. Budge: Experiences demoutrant que l'origine du nerf sympathique dans la moelle epiniere, Compt. rend. Acad. d. sc. **35**:255, 1851.

3. Bernard, Claude: Recherches experimentales sur le grand sympathique et specialment sur l'influence que le section de ce nerf exerce sur la chaleur animal, Compt. rend. Soc. de biol. **5**: Pt. 2, 277, 1853.

ocular phenomena resulting from stimulation and section of the cervical sympathetic, while Horner, in 1861, published the first clinical case of paralysis of this nerve. Mitchell, Moorehouse and Keene,[4] in charge of the nervous wards of the U. S. Army Hospital, Turner's Lane, Philadelphia, during the Civil War, gave the first accurate account of a gunshot wound of the sympathetic. Mr. Paget, in 1865, referred to this condition in connection with injuries to the brachial plexus.

Coming down to the present, Roche[5] reported four cases in 1916, and collected seven others, while Burger[6] recorded one in 1917, and Tournay[7] one in 1918. Our series of eleven cases was picked from 530 cases of peripheral nerve and spinal cord injury.

It is interesting to compare the prominence of symptoms in different patients, and the completeness or incompleteness of the ocular sympathetic syndrome. This may be due, according to Bernard,[8] to the fact that the center for oculopupillary action is situated at the eighth cervical and first dorsal vertebrae, while the centers for vasomotor, secretory and trophic changes are located at the second to sixth dorsal vertebrae. A lesion may thus involve one without affecting the others. In this connection, he demonstrated by experiments on dogs, that section of the first dorsal root produced oculopupillary symptoms, while section below this point of second to fourth roots caused only vasomotor changes.

Oppenheim demonstrated in a gunshot wound of the neck, where the first dorsal segment was exposed, that by stimulating the anterior roots oculopupillary symptoms were produced, but none occurred on stimulating roots below the first dorsal.

Krause thinks there are only oculopupillary symptoms if the spinal cord is affected, while there are vasomotor symptoms if the rami communicantes are principally involved. On the other hand, Mme. Dejerine Klumpke,[9] experimenting on dogs in 1885, demonstrated

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4. Mitchel, Moorehouse and Keene: Gunshot Wounds and Injuries of the Nerves, Philadelphia, J. B. Lippincott, 1864.

5. Roche: Les paralysies du sympathique cervical dans les blessures de guerre, Arch. d'Ophth. **35**:339 (Nov.-Dec.) 1916.

6. Burger: Contributions a l'etude de syndrome Claude Bernard-Horner, Arch. méd. Belges. **70**:309 (Apr.) 1917.

7. Tournay: Remarques sur l'inegalite pupillaire dans des cas de myosis unilateral par defect sympathique, Bull. de l'Acad. de méd., Par. **80**:486 (Dec. 3) 1918.

8. Bernard, Claude: Recherches experimentales sur le grand sympathique et specialment sur l'influence que le section de ce nerf exerce sur la chaleur animal, Compt. rend. Soc. de biol. **5**: Pt. 2, 277, 1853.

9. Klumpke, Mme. Dejerine: Dejerine's Semiologie des Affections du Systeme Nervous, Ed. 2, 1885, pp. 610 and 1156.

oculopupillary phenomena only when rami communicantes of the first dorsal nerve were affected.

It is not our purpose in this report to attempt to localize the pupillary, vasomotor or trophic fiber, but to present the cases in order of prominence of their ocular symptoms, and then to discuss them briefly from the standpoint of their neurologic diagnosis. Before doing this, however, a summary of the anatomy and physiology of the cervical sympathetic nerve might be of assistance.

ANATOMY AND PHYSIOLOGY OF CERVICAL SYMPATHETIC NERVE

The dilators and sphincters of the iris are too well known to need description, but the exact anatomy of the involuntary muscles of the orbits is still unsettled. In general, these muscles may be described as follows:

Behind the eyeball smooth muscle fibers occur in the periosteum and form a strong layer closing the orbital fissures. This is the musculus orbitalis which prevents the contents of the orbit from sinking backward. In the tendons of the levator palpebrae superiosis there are also smooth muscle fibers, and closely related to these are the tarsal muscles, small masses of involuntary muscle which lie at the bases of the upper and lower lids and insert into the tarsi. All of these, through their tonic activity, tend to keep the palpebral fissure widely open.[10]

The innervation of the pupil is double, the midbrain autonomic system sending fibers to the sphincter iridis, and the cervical sympathetic supplying the dilator. The orbital and tarsal muscles, and the nonstriated part of the levator palpebrae superioris are supplied only by the sympathetic system.

The spinal center for these sympathetic nerves lies in the lateral horn of the eighth cervical and first and second thoracic segments. Closely associated with this center are the cells of origin of the fibers that control the sweating and vasomotor reactions of the face. Some authors believe that the latter functions have their centers lower down the cord, in the second to fourth thoracic segments.[11] From these centers in the cord preganglionic fibers arise and pass via the corresponding cervical nerves and remi communicantes to the cervical sympathetic chain, finally terminating in the superior cervical ganglion.

Superimposed on the spinal center there is doubtless a cortical control, for such highly integrated reactions as fear and pleasure have their oculopupillary and vasomotor concommitants. A bulbar center also may be postulated, since lesions high in the cervical cord may cause myosis.

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10. Landstrom: Ueber Maebus Basedowi, Thesis, Stockholm, 1907.

11. Higier, H.: Nervous and Mental Diseases, Monograph Series No. 27, New York, 1919.

The peripheral nerves (in this case postganglionic sympathetic fibers) arise from cells in the superior cervical ganglion. These fibers at first are collected in the internal carotid plexus, but soon take separate courses. The more inferior branches probably spread out to supply the sweat glands and blood vessels of the face. The more superior branches generally follow the ophthalmic branch of the trigeminal nerve, and pass with the vasociliary nerve into the ciliary ganglion, from which they emerge and proceed with the long and short ciliary nerves to their endings in the smooth muscle fibers.

Physiologically, it must be kept in mind that the action of these nerves is tonic. In the case of the pupil there is a balance of two antagonistic systems, the autonomic sphincter and the sympathetic dilator; so the autonomic is allowed to act without restraint and cause continuous myosis if the sympathetic innervation is cut off. In the case of the vasomotor fibers to the blood vessels of the face, cutting off the vasoconstrictor control might be expected to cause flushing, but it is possible that there are also vasodilator fibers that may bring about active flushing. Here again the exact anatomy is not known so that the reactions cannot always be interpreted. Stimulation of the cervical sympathetic causes increased sweating of the face, so interruption of these fibers causes anhidrosis on the affected side. As the smooth muscles of the eyeball are normally in tonic contraction, they keep the globe forward and the lids separated. Cessation of this function is followed by ptosis, narrowing of the palpebral fissure and enophthalmos.

Due to the anatomic relations of the structures just described, certain symptoms are frequently associated with this sympathetic syndrome. In the first place, injuries to the spinal cord above or at the ciliospinal center may cause varying degrees of spastic paraplegia, quadriplegia or hemiplegia. Most common of all is the lower brachial plexus paralysis (Klumpke's paralysis) with atrophy of the small muscles of the hand and anesthesia of the dermatomes corresponding to the lowest cervical and upper two thoracic nerves. This condition indicates a lesion of the nerve roots before they enter the plexus. The recurrent laryngeal nerve may also be injured, causing hoarseness. In addition any number of traumas may be found, such as fractures of the vertebrae, injury to the larynx, etc. It is strange that any of the patients with these bullet wounds of the neck survived.

REPORT OF CASES

CASE 1.—*History.*—Private, Co. K, 30th Inf.; aged 23. Previous to the injury the history was unimportant. There was no suspicion of syphilis. Oct. 11, 1918, at Verdun, the patient was struck by a piece of shell in the left side of the neck just above the midclavicle; the missile lodged in the soft tissue just to the left

of, and slightly above, the spinous process of the third dorsal vertebra. There was immediate paralysis of the left arm, and the patient stated that for a week he saw double. By the first week in November he could move the shoulder, arm and forearm. Sensation also began to return except in the distribution of the eighth cervical root. At about this time the wound healed. Early in December voluntary motion returned at the wrist. He was admitted to U. S. Army General Hospital No. 11, on Nov. 30, 1918.

Examination.—Voluntary motion at the left elbow was weak and restricted. There was complete loss of flexion and extension of the fingers of the left

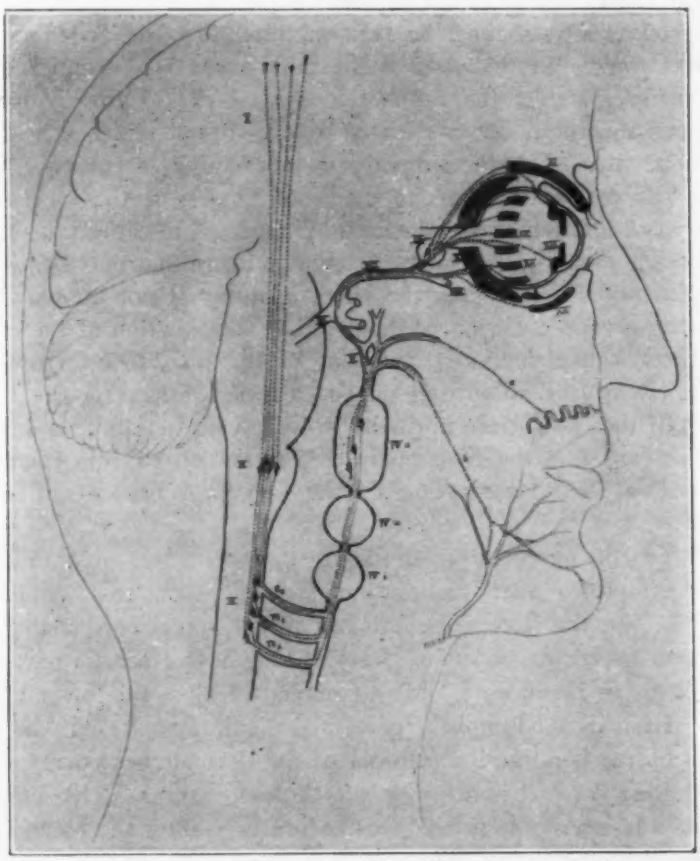

Fig. 1.—I, cortical path; II, bulbar center; III, ciliospinal center in lower cervical and upper thoracic cord, C. 8, Th. 1 and Th. 2 represent rami communicantes to cervical sympathetic chain; IV, cervical sympathetic ganglions, inferior, middle and superior; V, internal carotid plexus, giving off branches to (a) sweat glands of face, and (b) to blood vessels of face; VI, trigeminal nerve; VII, ophthalmic nerve; VIII, nasociliary nerve; IX, ciliary ganglion; X, orbital muscle (of Müller); XI, levator palpebral superioris with sympathetic branch to the smooth muscle fibers; XII, tarsal muscles; XIII, dilator muscles of the pupil. (N. B. The drawing is diagrammatic. No attempt is made to show the exact anatomic relations.)

hand, and only slight extension of the wrist. The entire extremity showed atrophy; the fingers were stiff and cyanotic. Sensory examination showed anesthesia in the distribution of the whole brachial plexus; but by January 23 this hypesthetic area was practically normal. With massage and electrical treatment there was marked improvement during the next three months, but no return of voluntary motion in the intrinsic muscles of the hand. Myosis, enophthalmos and ptosis of the left eye were noticed by the patient from the beginning. Unilateral anhidrosis was present on the left.

Diagnosis: Contusion of the whole left brachial plexus, with severance of the seventh and eighth cervical roots.

Ophthalmologic examination revealed marked enophthalmos, ptosis and myosis of the left side; left palpebral fissure 10 mm.; right, 13 mm; both pupils reacted to light, accommodation and consensually, but the left slower than the right. The diameter of the left pupil was 2.5 mm., right 3.5 mm.; under cocain, left 2.5 mm., right 5 mm. Tension, left eye 13 mm. of mercury, right 17 mm. of mercury; no heterochromia irides. Vision 20/20 in both eyes; near point left 11 cm., right 12 cm. Ocular movements normal. Left globe slightly elevated and left nictitating membrane slightly more prominent than right. Left cornea appeared a little flatter than right. On excursion of the eye to the right, the

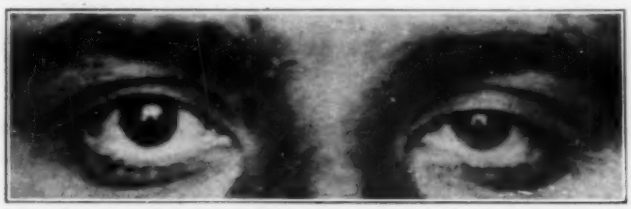

Fig. 2 (Case 1).—Marked enophthalmos, ptosis and myosis of the left eye.

difference in the size of the pupils increased slightly, while on turning to the left the difference diminished slightly. Fundi and fields were normal. No flushing of the face or hemiatrophy was noted. Lacrimation was normal.

Case 2.—*History.*—Private, Co. L, 18th Inf.; aged 24. The past history was negative. He did not have syphilis. At Soissons, July 21, 1918, he was struck by a piece of high explosive shell in the right forearm and was thrown about 5 feet by the explosion, landing on his right shoulder. The right arm immediately became numb and paralyzed. At Evacuation Hospital No. 5, he was operated on and the bones of the right arm were set. No ether was necessary on account of the anesthesia of the arm. During the next three months there was no improvement in the paralysis. He was admitted to U. S. Army General Hospital No. 11, Oct. 24, 1918.

Examination.—This revealed that on the outer side of the right forearm there was a scar 1.5 inches long. The right arm was completely paralyzed, and there was restricted motion of the shoulder. Electrical examination shows normal response to faradic stimulation in the trapezius and supraspinatus and infraspinatus muscles; all the other muscles of the right upper extremity failed to respond. With the galvanic current there was a slow wavy contraction in all the muscles that failed to respond to the faradic, except the intrinsic muscles of the anterior surface of the hand in which there was no response. Sensory examination showed anesthesia in the distribution of the fifth, sixth, seventh and eighth cervical and first thoracic nerves.

On Nov. 14, 1918, an operation was performed by Lieut.-Col. Charles H. Frazier. The posterior triangle of the neck on the right was explored and a bulbus enlargement was found that represented the remnants of the brachial plexus. It extended from the clavicle upward, tapering off into a single trunk which proved to be the fifth cervical. The retracted stumps of the sixth, seventh and eighth cervical nerves could not be found, although search was made almost up to the intervertebral foramina. After this operation the patient stated he first noticed that his right pupil was smaller than the left. It is probable that the

Fig. 3 (Case 2).—Inequality of pupils before instillation of cocain.

oculopupillary symptoms were present immediately after the injury, but passed unnoticed; and that their onset had no relation to the operation. A second operation was performed on Dec. 19, 1918, by Lieut.-Col. Frazier. The exposure was similar and the fifth cervical nerve root was identified and grafted with two strands of the musculocutaneous nerve to a nerve in the shoulder, which was thought to be the circumflex.

Diagnosis. Evulsion of the sixth, seventh and eighth cervical nerve roots and of the first thoracic root of the right brachial plexus with contusion and fibrosis of the fifth cervical root.

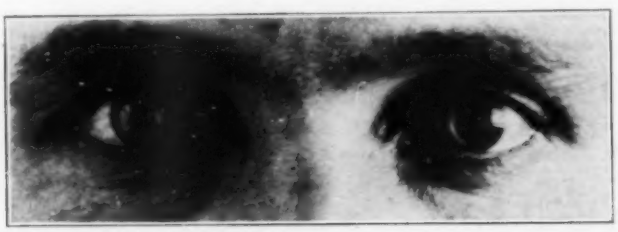

Fig. 4 (Case 2).—Showing increase of inequality after instillation of cocain.

Ophthalmologic Examination.—This revealed marked enophthalmos, ptosis and myosis of the right side; right palpebral aperture 9 mm., left 12 mm.; pupillary reactions normal, but right slightly retarded; diameter right pupil 2.5 mm., left 6 mm.; width under cocain, right 2.5 mm., left 9 mm.; ocular tension, right 12 mm. of mercury, left 16 mm. of mercury; vision 20/15 both eyes. Near point, right 9 cm., left 10.5 cm.; right eye slightly elevated; ocular movements normal. On excursion of the eyes to the right the difference in the size of the pupils diminished, on excursion to the left the difference in the size increased slightly. Fundi and fields were normal. The patient showed a slight hemiatrophy of the right side of the face.

CASE 3.—*History.*—Private, Co. C, 58th Inf.; aged 27. The past history was negative. On October 5 he was struck by a piece of shrapnel while advancing at Verdun. This missile entered in the midline anteriorly just below the larynx and passed out of the lateral aspect of the neck through the trapezius muscle. There was immediate paralysis of the left arm and aphonia. Two weeks later his voice and the movements of the left hand began to return. A week later he noticed that his left eye was sensitive and that the lashes fell out. From this time on there was steady improvement. He was admitted to U. S. Army General Hospital No. 11 on Dec. 31, 1918.

Examination.—Physical examination showed paralysis of the left deltoid and supraspinatus and infraspinatus muscles, with weakness of the brachicardialis, biceps and triceps. There was no sensory disturbance. The voice was hoarse and laryngoscopy showed partial paralysis of the left vocal cord. The left palpebral fissure was narrower than the right and the pupil contracted. During the next four months there was improvement in the voice and the arm movement, but the scapular muscles and deltoid were still atrophied, and weak.

Diagnosis: (1) Contusion of left brachial plexus with a severe lesion of the fifth cervical root, causing a more permanent scapulohumeral type of paralysis; (2) partial paralysis of left recurrent laryngeal nerve; (3) interruption of left cervical sympathetic nerve.

Ophthalmologic Examination.—Marked enophthalmos, ptosis and myosis of left eye. Left palpebral aperture 7 mm., right 10 mm. The pupils reacted normally, the left being slightly slower than the right; width of left pupil 2.5 mm., right 3.5 mm.; under cocain, left 2.5 mm., right 5 mm.; tension of left eyeball 23 mm. of mercury, right 25 mm. of mercury. The left cornea appeared slightly flatter than the right. The vision of the right eye was 20/20, left 20/20; near point, left 11 cm., right 13 cm. Ocular movements were normal. The left eye was slightly elevated. There was slight diminution of relative size of pupils on gazing to extreme left, with a slight increase in size on directing gaze to the right. Fundi and fields were normal. There was no disturbance of lacrimation or of vasomotor functions.

CASE 4.—*History.*—Private, Co. D, 47th Inf.; aged 30. The past history was negative. No syphilis was present. On July 30, on the Champagne front, he was struck three times—first by a piece of shrapnel in the neck entering just to the left of the thyroid cartilage and passing out behind the sternomastoid muscle at the same level; second, by a machine-gun bullet from behind, which fractured the first thoracic vertebra and lodged in the soft parts, being removed the next day; and third, he received a gutter wound in the biceps of the right arm. His legs were stiff and weak, and he was unable to walk. He could not speak and the left arm was paralyzed. During September his voice returned, but was hoarse. In late October he could walk a few steps if supported.

Examination.—Examination on admission to U. S. Army General Hospital No. 11 revealed that the whole right side of the body below the third rib was anesthetic to pain and temperature stimuli. In the distribution of the eighth cervical and first and second thoracic nerves on the left there was anesthesia to all forms of cutaneous stimulation. The arm was carried partly flexed, and motion was limited at wrist and elbow; the thumb and fingers were spastic and weak with fibrosis of the joints. The muscles of the trunk were hypertonic and the tendon reflexes were greatly exaggerated with bilateral clonus. Babinski's sign was elicited on both sides by touching the patient anywhere below the third rib, and even by jarring the bed. Electrical examination showed that all muscles

reacted to the faradic current. There was hyperhidrosis of the right side of face. The left eye showed ptosis, enophthalmos and myosis. Roentgen-ray examination revealed a fracture of the first thoracic vertebra.

Course and Treatment.—On November 5 laminectomy was performed by Lieut.-Col. C. H. Frazier, and the cord was decompressed at the eighth cervical and first and second thoracic vertebrae.

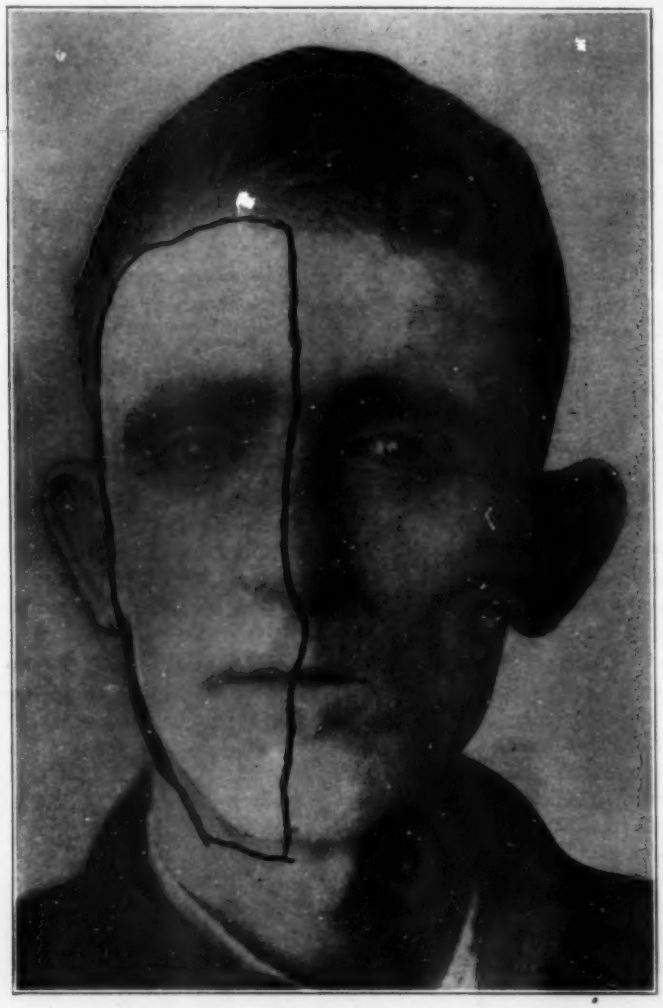

Fig. 5 (Case 4).—Showing unilateral sweating of the face.

Three months later the patient was able to walk half a mile. Anesthesia on the right had disappeared, but it was still present in the left arm. The oculopupillary and sudomotor symptoms persisted. The tendon reflexes were still exaggerated, and Babinski's sign could be elicited on both sides, but the clonus had disappeared. The left arm was more freely moveable, but the fingers were still spastic and weak.

Diagnosis: (1) Contusion of the spinal cord at the level of the eighth cervical and first and second thoracic segments, from machine-gun bullet. (2) Interruption of the left recurrent laryngeal nerve, and of the left cervical sympathetic nerve by a piece of shrapnel.

Ophthalmologic Examination.—This revealed enophthalmos, ptosis and myosis of left side, less marked than in previous cases; left palpebral fissure 11 mm., right 13 mm.; pupillary reactions normal, but left slower than right; diameter left pupil 2.5 mm., right 3.5 mm.; under cocain, left 2.5 mm., and right 4.5 mm. Tension was the same in both eyes. Vision was 20/20 in both eyes. Ocular movements were normal in all directions. No change was noted in the relative size of the pupils when looking to the right or the left. The fundi were normal. There was no disturbance in lacrimation; but marked flushing on the left side of the face.

CASE 5.—*History.*—Corporal, Co. F, 26th Inf.; aged 20. The past history was negative; there was no history of syphilis. At Soissons on July 19, 1918, the patient was struck in the right side of the neck by a machine-gun bullet that entered 2 cm. above and 4 cm. to the right of the thyroid cartilage and lodged to the right of the vertebral column at the level of the fifth and sixth cervical vertebrae. Four days later it was removed at operation. There was immediate paralysis of the right arm and shoulder, which began to improve after a few weeks.

Examination.—On admission to U. S. Army General Hospital No. 11, on August 27, motor disability was limited to weakness of the right arm and hand, with inability to flex the thumb and index finger. There were sensory disturbances in the area of distribution of the seventh root; light touch and pain were poorly felt, and there was pain and tenderness in the radial half of the forearm and hand. Electrical examination showed that all muscles responded to the faradic current. Narrowing of the right palpebral fissure, with enophthalmos and myosis was conspicuous.

During October there was marked improvement, and late in December he was transferred to a Convalescent Detachment.

A special eye examination, December 10, showed: Vision, right 20/15, left 20/15. External examination: Slight ptosis of right upper lid. Right pupil 3 mm. in diameter, left 4.5 mm., both reacted to light and accommodation. Ocular movements were good in all directions. Ophthalmoscopy: Fundi negative.

Diagnosis: (1) Contusion of the right brachial plexus causing temporary complete paralysis of the right arm, with a residual irritative lesion of the seventh cervical root, causing symptoms for six months; (2) interruption of cervical sympathetic nerve in the neck.

Ophthalmologic Examination.—This revealed enophthalmos, ptosis and myosis of right side. Width of right palpebral fissure was 8 mm., left 10 mm. Pupils reacted to light and accommodation. Diameter of right pupil was 3 mm., left 4.5 mm.; under cocain, right 3 mm., left 7 mm. Ocular movements were good in all directions; fundi negative; vision, right 20/15, left 20/15. There was unilateral anhidrosis on right side.

CASE 6.—*History.*—Private, Co. G, 28th Inf.; aged 20. The past history does not reveal syphilis or other important facts. On July 18 he was struck in the right posterolateral aspect of the neck by a machine-gun bullet which passed forward and downward, coming out through the thyroid cartilage slightly to the right of the midline. There was immediate paralysis of this shoulder and upper arm, with aphonia for two weeks, and hoarseness thereafter. On August

21 the oculopupillary symptoms were noted. On August 27 there was no return of motor function and hypesthesia was found on the right arm in the distribution of the seventh cervical nerve. On October 3 he was admitted to U. S. Army General Hospital No. 11.

Examination.—The examination here showed paralysis and atrophy of the right scapulohumeral muscles, with marked atrophy and no response to faradic stimulation, except weak contractions in the triceps and biceps. There was no sensory disturbance. Myosis, enophthalmos and ptosis were present on the right, with unilateral anhidrosis. There was gradual improvement, and on February 13 he was discharged with voluntary movement and faradic response in all muscles. The oculopupillary symptoms did not improve.

Diagnosis: (1) Contusion of the outer cord of the right brachial plexus causing paralysis of the right arm and shoulder of the scapulohumeral type; (2) interruption of the right cervical sympathetic nerve in the neck; (3) interruption of the right recurrent laryngeal nerve.

Ophthalmologic Examination.—Enophthalmos, ptosis and myosis were present on the right. The right palpebral aperture was 6.5 mm., left 8.5 mm. Pupillary reactions were normal; pupillary measurements not recorded; vision, right 20/50, left 20/20. Refractive error was present in the right eye; fundi negative; unilateral anhidrosis.

Case 7.—*History.*—Private, M. G. B., 9th Inf.; aged 28. On July 1, 1918, at Chateau Thierry, he was wounded by shrapnel in the neck and right arm. The projectile entered the neck 5 cm. above the junction of the inner and middle thirds of the clavicle and lodged against the spinal column, fracturing the transverse process of the seventh cervical vertebra.

For the first month he had pain in both arms, paralysis of both legs, and of the right arm, and weakness of the left arm. Bladder and rectal control were poor for six weeks. There was steady improvement and by March 6, when he was admitted to U. S. Army General Hospital No. 11, he could walk stiffly.

Examination.—The examination showed a Brown-Séquard paralysis with loss of pain and temperature sense on the left side below the seventh cervical segment, and weakness and spasticity of the right leg. There was also a partial musculospiral nerve lesion of the right arm. Myosis of the right pupil and "hemisweating" were noted on July 6. At operation on April 15, 1919, a fragment of shell was removed from the right trapezius muscle.

Diagnosis: Contusion of right side of the spinal cord at the level of the eighth cervical segment.

Ophthalmologic Examination.—This revealed enophthalmos, ptosis and myosis of the right side; width of right palpebral fissure 10.5 mm., left 12 mm. The pupils reacted normally to light and accommodation; size of right pupil 2.5 mm., left 3 mm. Under cocain, right 3.5 mm., left 6 mm. Ocular tension, right 21 mm. of mercury, left 23 mm. of mercury; vision, right 20/20, left 20/20. Muscular excursions were normal. On turning the eyes to the extreme right there occurred a slight diminution in difference between the size of the two pupils, while the opposite occurred when looking to the extreme left; fundi negative. There was anhidrosis of the right side of the face.

Case 8.—*History.*—Private, Co. B, 11th M. G. B.; aged 29. The past history was negative. Syphilis was denied by name and symptom. On Sept. 28, 1918, while on duty in the Argonne Forest, he was struck by a piece of aerial bomb, which entered the neck in the midline anteriorly and did not emerge. The left arm was immediately paralyzed completely and the legs were weak.

There was also bleeding from the mouth and inability to speak. A week later voluntary motion began to return in the fingers, and since that time there had been steady improvement. The legs regained their motor power in two months. There was difficulty in controlling the bladder for two months. No eye symptoms were noticed by the patient. He was in bed most of the time until his admission to U. S. Army General Hospital No. 11, on Dec. 5, 1918. He could then walk a short distance, and by January 1 could walk a mile.

Examination.—Physical examinations made at various times from December 5 until March 17 and in various special departments revealed: Tachycardia constant between 110 and 130; the heart was otherwise normal. There was a small circular wound scar on the anterior surface of the neck in the midline over the larynx. Voluntary motion was weak at the left shoulder, elbow and wrist. There was atrophy of the left arm, forearm and hand. The skin of this forearm and hand showed scaliness and the nails were long and tender. There was hypohidrosis in the left hand. The tendon reflexes were exaggerated on the left, and in the left leg there was a short patellar clonus and a positive Babinski sign. The whole right side of the body showed a hypesthesia to pain and temperature below the level of the tenth dorsal segment. The left vocal cord was fixed near the midline. The left palpebral fissure was narrower than the right. There was pain in the left shoulder and hand, and the muscles of the left arm were tender, especially the triceps.

Diagnosis: (1) Contusion of the spinal cord in the region of the eighth cervical segment, more on the left than on the right side; (2) contusion of the sixth, seventh and eighth roots; (3) interruption of left recurrent laryngeal nerve.

Ophthalmologic Examination.—This revealed enophthalmos, ptosis and myosis of the left eye; left palpebral fissure 9 mm., right 11 mm. Pupils reacted to light and accommodation, but the left was slightly slower than the right. Diameter of the left pupil was 3 mm., right 3.5 mm.; under cocain, left 5.5 mm., right 7 mm.; ocular tension, left 16 mm. of mercury, right 17 mm. of mercury; vision, left 20/20, right 20/20 uncorrected; left near point 12 cm., right 16 cm. No abnormal change in the relative size of the pupils was apparent on turning the eyes to the extreme right or left. The left nictitating membrane appeared slightly more prominent than the right. Fundi and fields were negative. Slight diminution of sudomotor activity was noted on the left side of the face.

Case 9.—*History.*—Private, Battery I, 6th Field Artillery; aged 31. The past history was negative—no syphilis. On June 4, 1918, at Cantigney, the patient was struck in the back of the neck with a piece of high explosive shell that fractured the sixth cervical vertebra and lodged in the vertebral canal. He was immediately completely paralyzed in all four extremities. About July 15 voluntary movement began to return in both hands and arms, and a month later in the left leg. There was no movement in the right leg until November. There was complete loss of bladder and rectal control until October, when he began to have slight improvement. He was admitted to U. S. Army General Hospital No. 11, on Nov. 6, 1918.

Examination.—Physical examination showed hypalgesia below the second rib on the entire left side. The right palpebral fissure was narrower than the left. There was motor weakness and spasticity in all four extremities; the movement of the right leg was limited to flexion of the toes, the right arm was weaker than the left.

Course and Treatment.—Treatment was directed to overcome spastic contractures and decubital ulcers. On January 23 a laminectomy was performed by Lieut.-Col. Charles H. Frazier, and a fragment of shell was removed from the dura at the level of the junction of the sixth and seventh cervical vertebrae; it was pressing on the right posterior aspect of the cord. In the next three months there was slight improvement in the movement of the legs, due largely to physiotherapy, but the patient was still in bed with bad contractures of the legs. Bladder control improved immediately after operation so that he could hold his urine for nearly two hours, instead of fifteen minutes as was the case just before operation.

Diagnosis: Compression of the seventh and eighth cervical segments of the cord, mainly on the right side, causing a partial Brown-Séquard syndrome and narrowing of the right palpebral fissure.

Ophthalmologic Examination.—This revealed enophthalmos, ptosis and myosis on the right side. Right palpebral aperture was 10 mm., left 11.5 mm. The pupils reacted to light, accommodation and consensually. Slight difference in activity between the two was noted, the right being a little slower than the left. Diameter of the right pupil was 3.5 mm., left 4 mm.; under cocain, right 4.5 mm., left 8 mm.; tension, right eye 18 mm. of mercury, left 22 mm. of mercury; vision, right eye 20/20, left eye 20/20; right near point 10 cm., left near point 12 cm. The right eye was slightly elevated from the normal position. The fundi were normal.

Case 10.—*History.*—Private, Co. I, 9th Inf. The past history was negative, and there was no evidence of syphilis. On July 18, 1918, at Soissons, the patient was struck by a machine-gun bullet. At the time he was aiming at a machine-gun nest in a tree, and he thought the bullet came from that gun and struck him in the left cheek, ranged downward and came out just at the left of the spinal column. He was immediately paralyzed in all four extremities, but in two weeks he could move his legs and right arm freely. During the next four months there was steady improvement, and in November he could walk a mile.

Examination.—In April, 1919, the positive findings were: Fracture of the fifth, sixth and seventh cervical vertebrae shown by the roentgen ray; stiffness and limitation of motion in left hand and wrist; tendon reflexes increased in arms and legs, less in left arm than in other extremities; muscular spasms in arms and legs occasionally. Constipation was present; there was no bladder disturbance, but there had been loss of libido and potentia since the injury.

Diagnosis: Contusion of the spinal cord in the region of the seventh and eighth cervical segments.

Ophthalmologic Examination.—Enophthalmos and myosis were present on the left side. No ptosis was noted. The width of each palpebral fissure was 10 mm. The diameter of the left pupil was 3.5 mm., right pupil 5 mm. The pupils reacted to light, accommodation and consensually; activity of the left was slightly diminished. Tension of the left eye was 20 mm. of mercury, right eye 25 mm. of mercury; vision, left eye 20/20, right 20/20; near point of left eye 13 cm., of right eye 14 cm. Muscular excursions were normal; fundi normal.

Case 11.—*History.*—Corporal, Co. E, 125th Inf.; aged 23. On Oct. 5, 1918, he was struck by a machine-gun bullet which grazed the right shoulder and passed into the right side of the neck and out of the left side of the neck at a slightly higher level. Aphonia was the only symptom noticed immediately, and after a few weeks he was able to whisper hoarsely.

Examination.—Examination, made November 5, showed immobility of the right vocal cord. On Feb. 7, 1919, this cord moved slightly and the voice was

less hoarse. A slight narrowing of the left palpebral fissure was present; there was no myosis or unilateral sweating. (See special ophthalmologic examination.) Routine neurologic examination elicited no spinal cord symtoms. He was discharged on February 27, with improvement in his voice.

Diagnosis: (1) Partial paralysis of the right recurrent laryngeal nerve, recovering; (2) partial interruption of the left cevical sympathetic nerve. This diagnosis seems probable, because the symptoms do not correspond to those found either in the group of cord contusions or of nerve lesions presumably complete.

Ophthalmologic Examination.—Enophthalmos, ptosis and slight narrowing of palpebral fissure were noted on the left side. No myosis was present. The left palpebral aperture was 11 mm., the right 12 mm. Both pupils reacted normally to light and accommodation. Each measured 3 mm. in diameter, and under cocain the dilatation was the same on both sides. Vision in both eyes was 20/20. Ocular movements were normal in all directions. The fundi were negative.

FINDINGS AND DIAGNOSIS IN AUTHORS' CASES

Case	Enophthalmos	Ptosis	Myosis	Hypotension	Palpebral Fissure	Pupil	Pupil (Cocain)	Vision	Near Point	Unilateral Anhidrosis	Vasomotor Disturbance	Hemiatrophy Facialis	Heterochromia Irides	Diagnosis
1	+++	++	+	+	13 10	3½ 2½	5 2½	20/20 20/20	12 11	+	—	—	—	R. Root severance 7, 8 cervical
2	+++	++	+	+	9 12	2½ 6	2½ 9	20/15 20/15	9 10½	+	+	+	—	R. Root evulsion 6, 7, 8 cerv. 1st th.
3	+++	++	+	+	13 10	3½ 2½	5 2½	20/20 20/20	13 11	—	—	—	—	Interruption of L. cerv. sympathetic nerve
4	+	+	+	—	13 11	3½ 2½	4½ 2½	20/20 20/20	12 11	+	+	—	—	Interruption of L. cerv. sympathetic nerve
5	+	+	+	—	8 10	3 4½	3 7	20/15 20/15	..	+	—	—	—	Interruption of R. cerv. sympathetic nerve
6	+	+	+	—	6½ 8½	.. ..	.. ..	20/50 20/20	..	+	—	—	—	Interruption of R. cerv. sympathetic nerve
7	+	++	+	+	10½ 12	2½ 3	3½ 6	20/20 20/20	9 9	+	—	—	—	R. cord contusion, C. 8
8	++	+	+	—	11 9	3½ 3	7 5½	20/15 20/20	16 12	—	—	—	—	L. cord contusion, C. 8
9	+	+	+	+	10 11½	3½ 4	4½ 8	20/20 20/20	10 12	—	—	—	—	R. cord compression, C. 7, 8
10	+	—	+	+	10 10	5 3½	7 5	20/20 20/20	14 11	—	—	—	—	L. cord contusion, C. 7, 8
11	+	+	—	—	12 11	3 3	.. ..	20/20 20/20	10 10	—	—	—	—	Partial interruption of L. cerv. sympathetic nerve

SUMMARY

In correlating the above data, the most striking fact is shown by arranging the cases in the order of the severity of their ocular symptoms, and then observing the grouping of the neurologic diagnoses.

Cases 1 and 2, the most severe, are both root lesions. They are followed by four cases of lesions of the cervical sympathetic nerve. All of these showed more conspicuous eye phenomena than the following four cord contusions. The last case had mild symptoms and seemed to be a nerve lesion, but the neurologic examination was unsatisfactory, and there is a possibility that this case was one of cord contusion, or partial interruption of the cervical sympathetic nerve.

Reviewing separately the more important symptoms, these results are presented:

1. Enophthalmos was the most constant symptom, being present in all cases, but most marked in the two root lesions, with a diminishing degree of prominence as we go down the scale through the nerve injuries, until the cord contusions are reached, where in Cases 10 and 11 it was only faintly perceptible.

2. Only one case failed to show at least a moderate degree of ptosis, and this was one of cord contusion.

3. Miosis was present in all except Case 10 (the neurologic examination of which, as we above mentioned, was unsatisfactory). It is interesting that the pupils of the cases with the more severe lesions, namely the root and nerve injuries, failed to dilate under cocain, while the cord contusions dilated, but to a reduced degree, the range being from 1 to 2 mm.

4. Hypotension was present in only five of eleven cases — the two root and three contusion injuries. The nerve lesions showed no difference in tension.

5. In comparing the width of the palpebral apertures, we find that the greatest difference between the sound and affected sides is seen in Cases 1 and 2 (root lesions) and Case 3 (the most severe nerve injury). Each of these shows a difference of 3 mm. The other nerve cases register a difference of 2 mm., while three of the four cord lesions present a variance of 1.5 to 2 mm.

6. Sudomotor disturbances were recorded in over 50 per cent. of our cases, and as with the other symptoms, unilateral anhidrosis was present in the more severe lesions, namely, Cases 1, 2, 4, 5, 6 and 7.

7. Only two patients showed vasomotor disturbances, Cases 2 and 4. The explanation for this may be that we did not see our patients until four to six months after injury, and possibly symptoms that existed immediately afterward had disappeared.

8. Heterochromia irides was not present in any one of the eleven cases examined. Presuming that this condition is due to paralysis of the cervical sympathetic nerve, this symptom might be expected in the cases classified under root lesions, and the more severe nerve injuries, in which complete interruption was undoubtedly present.

Wilson [12] reported a case in 1917 in a girl 14 years of age, whose condition had existed for twelve years, resulting from swollen cervical glands. Calhoun [13] has recently reported four cases, and found only thirty-three others recorded in the literature. In all of these, heterochromia had existed since infancy or youth. After an exhaustive study, he says that heterochromia irides has never been seen in adults, as the result of sympathetic paralysis from trauma. Bistis [14] thus far has reported the only case occurring in adults, following paralysis of the cervical sympathetic nerve. As all the cases here reported were adults, in whom the paralysis had been caused by gunshot wounds, the statement of Calhoun seems to explain the absence of heterochromia irides in this series.

9. Hemiatrophy facialis was observed to a slight degree in one patient, Case 2, a root evulsion, involving the sixth, seventh and eighth cervical and first thoracic roots. Weisenberg, in an unreported case seen in U. S. Army General Hospital No. 3, saw hemiatrophy in a similar wound, six months after injury, but as in the above, it was not pronounced. Possibly if we were to observe these patients several months longer, this condition would develop in a larger number, as it has been observed quite constantly in paralysis of the cervical sympathetic of long standing.

10. Vision was normal or better in all patients but one, in whom there was a refractive error corrected to normal. An interesting point was noted in the difference between the near points of the good and affected sides. Nearly all showed a variation of from 1 to 4 cm.

11. According to Tournay,[15] the relative difference in size between the miotic and normal pupils diminishes on directing the eyes to the affected side, while the opposite is true when the eyes are turned toward the healthy side. This we found to be true to a slight degree in a few cases, but it was not a constant symptom.

CONCLUSIONS

1. Lesions affecting the seventh and eighth cervical and first thoracic roots, through which sympathetic fibers run, cause the most severe and typical oculopupillary syndromes.

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12. Wilson: Heterochromia Irides in Nerve Case, J. Nerv. & Ment. Dis. **1**:438, 1917.

13. Calhoun: Causes of Heterochromia Irides with Special Reference to Paralysis of the Cervical Sympathetic, Am. J. Ophth. **2**:255 (April) 1919.

14. Bistis: La paralysie du sympathique dans l'etiologie de l'heterochromie, Arch. Ophth. **32**:578, 1912.

15. Tournay: Remarques sur l'inegalite pupillaire dans des cas myosis unilateral par defect sympathique, Bull. de l'Acad. de méd. Par. **80**:486 (Dec. 3) 1918.

2. Lesions involving the cervical sympathetic nerves, with complete or partial interruption, produce symptoms less severe, as a rule, than root injuries.

3. Contusions of the cord result in the least pronounced phenomena, and are more prone to complete recovery than the first two conditions.

4. Heterochromia irides did not occur in adults following injury to the cervical sympathetic nerve.

5. The pupils of the cases with root and nerve injuries did not dilate with cocain; those of the cord contusions did.

6. The near point of the affected eye was less than its fellow in over 50 per cent. of the cases, the average difference being slightly over 2 cm. It is difficult to draw any definite conclusion on this point, as the greatest variation was in a case of cord contusion, with only slight ocular symptoms.

Explanation of Plate I

Fig. 1 (Case 1).—Arrow shows point at which shell struck neck.

Fig. 2 (Case 2).—Arrow shows point at which blow was received when patient landed on right shoulder. Shaded portion shows area of sensory disturbance.

Fig. 3 (Case 3).—Arrow shows point of entrance and exit of missile.

Fig. 4 (Case 4).—Arrows show path of shrapnel and point of entrance of machine-gun bullet at first thoracic vertebrae. Shaded portions are areas of sensory disturbance.

Fig. 5 (Case 5).—Arrow shows point of entrance of machine-gun bullet near thyroid cartilage. Shaded portion is the area of sensory disturbance.

Fig. 6 (Case 6).—Arrow shows path of machine-gun bullet. Shaded portion shows area of sensory disturbance.

Fig. 7 (Case 7).—Arrow shows point of entrance of shrapnel. Shaded portion shows area of sensory disturbance.

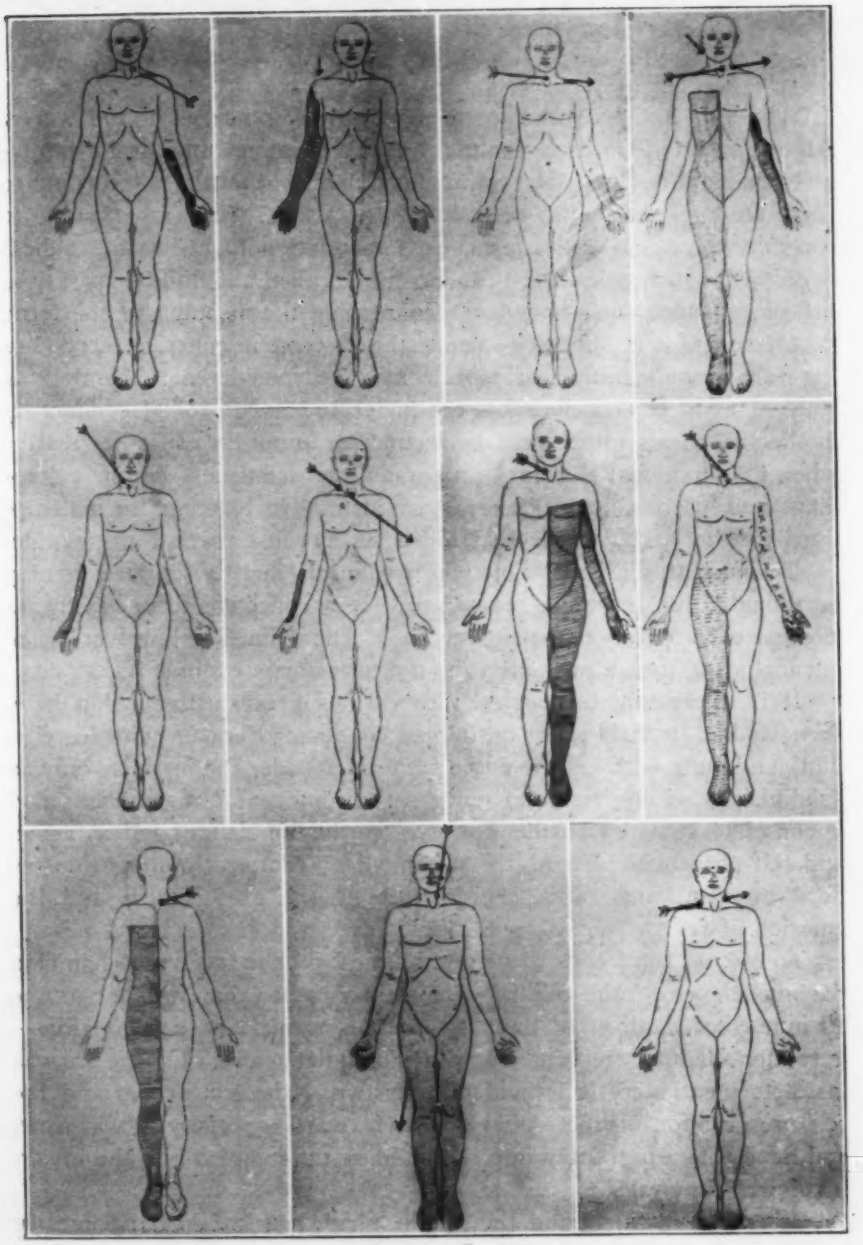
Fig. 8 (Case 8).—Arrow shows point of entrance of aerial bomb. Crosses on left arm indicate area of atrophy; shaded portion shows area of hypesthesia to pain and temperature.

Fig. 9 (Case 9).—Arrow shows point in neck at which high explosive shell struck. Shaded portion is area of hypalgesia.

Fig. 10 (Case 10).—Arrow shows points of entrance and exit of machine-gun bullet.

Fig. 11 (Case 11).—Arrow shows path of machine-gun bullet.

PLATE 1



MEDITATIONS ON MORALE*

DONALD GREGG, M.D.

WELLESLEY, MASS.

The war has produced many new words or usages of old words. Among such old words is one that has taken on meanings and a familiarity that did not exist in prewar days, namely, morale. According to former usage, morale has been defined as a state of mind pertaining to courage, valor and enthusiasm, used in relation to bodies of soldiers or sailors. Such a definition can hardly be called a definition, as it does little to advance one's knowledge concerning the meaning of the term.

Of course it is difficult to define the abstract in concrete terms, but the following definition of morale may be considered. Morale is a state of mind or consciousness conducive to the control of conduct by intellectual ideals rather than by instinctive impulses. In other words, when the emotional element in a person's consciousness becomes dominant and his conduct is governed by instinctive reactions to his emotions instead of by intellectual ideals he has lost part of his morale.

To some this definition may prove unsatisfactory, because it may be thought that morale is more properly a collective term having an element of cooperation for a given end. The definition, however, without including such a meaning does not necessarily exclude it.

It is interesting to consider the various great instincts that have been studied in their effect on human conduct. Bergson with his élan vital, and Jung with his life force seem to consider the instincts only as subdivisions of one essential power—the source of our conscious and unconscious strivings. Adler has stressed the impulses of self-assertion and self-abasement; Freud, the sex impulse; Trotter, the gregarious or herd impulse; and Sidis, the instinct of self-preservation and the emotion of fear.

To discuss the trends of modern clinical psychiatry, or to champion the importance of the rôle of one instinct rather than another as the all important catalizer of human conduct—normal and psychopathic—is to step off into deep and by no means quiet water. Of the various instincts mentioned, it is evident that two — the gregarious and the self-preservation instincts—have come forward especially in war time, and have entered strongly into the building up and also into the breaking down of morale.

When the instincts and the intellect have worked in harmony, we have usually found mental health, activity and euphoria; when the

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* Read at the October meeting of the Boston Society of Psychiatry and Neurology.

instincts and the intellect have been in conflict, we have seen cacophoria, hesitation and often ill health. Should we buy liberty bonds or pay our debts; should we stick to our home job or enlist? The conflict of the herd instinct calling us to do as our colleagues were doing, and the intellectual decision to do something else, is still vivid in our minds, and last November marked the end of more than one conflict.

The factors conducive to the augmentation of the instinctive reactions may be considered under three heads: (1) inheritance, (2) acquired disabilities, and (3) environmental strain.

Inheritance influences morale along one or both of two lines. In the first place, unquestionably there is of course vast variation in congenital physical conformation and development, and strength along the more subtle physical characteristics that are defined as immunity to disease. Some persons are handicapped by being born bleeders; some persons, on account of their physical conformation, are predisposed to certain mechanical difficulties such as malnutrition from a sagging stomach or headache from focusing eyes that are inclined to squint. Others are poorly started in life—they are premature infants or are born of sick mothers. Such congenital physical disabilities unquestionably increase the burden borne by many in their struggle to live successful and happy lives. Such disabilities, however, by no means necessarily lessen morale. In many cases the person by a sort of compensatory process develops exceptional ability along unhandicapped lines and makes a resulting success of life which often surpasses that of the physically well endowed.

Another way in which congenital handicaps may lessen morale is through fear of following in the footsteps of parents, which predisposes to the development of ills and handicaps from which the parents suffered. Many an individual explains his bad temper on the ground that his father had a bad temper, or attributes his indulgence in alcohol to a similar parental weakness, or in explanation of a headache says that his mother had the same kind of headache. If mental disease has shown itself in a parent, a patient may be under constant dread of the appearance of a similar condition in himself. Dread of developing weaknesses and conditions exhibited by a previous generation undoubtedly is often an important factor in the inception of a conscious or subconscious fear, and such a fear is a definite source of lowered morale.

Acquired disabilities either of a temporary or permanent character tend to reduce morale. They do this in the first place by reducing physical strength, and thus increasing the difficulties that must be surmounted to gain success. The greater the difficulty with which success is attained, the more likely an individual is to fear failure in the attainment of success; or, in other words, the more easily is the instinct

of self-preservation and its accompanying fear, emotion, aroused. In the second place, acquired physical disability may render a person more sensitive to emotional strain and also more predisposed to react excessively to such emotional strain. These excessive reactions gain the attention and in turn become secondary objects of fear and apprehension.

Acquired disabilities, especially if permanent or of long standing, may cease to be a cause of lowered morale and the person may develop along unhandicapped lines exceptional ability tending to unusual success.

Environmental strain caused many of the war neuroses, and here, as many writers have pointed out, the instinct of self-preservation and the emotion of fear have been the most important factors.

The work of the morale division of the army has been mainly to foster and augment intellectual ideals, and to remove or mitigate as far as possible the conditions that beget ill health, anxiety and fear. It has extolled the reasons for the entry of America into the war; it has encouraged self-confidence and developed esprit de corps, furthered pride in the various army divisions and preached the invincibility of American troops. It has encouraged every agency that combated discomfort and homesickness and has glorified the importance and necessity of personal purity. Poor food, poor shelter, lack of occupation, poor organization, petty rivalries and jealousies among men and officers and physical illness—both venereal and nonvenereal—lessened morale. Doughnuts, songs, sports, letters from home, decorations for valor and other encouragements, by lessening emotional strain and augmenting intellectual ideals helped keep the morale high.

If we could plot out on a chart our intellectual ideals and our instincts, would we not find that our morale would be represented by the separation between the two curves? With fall in ideals, or with a rise of instinctive reactions, the margin of morale decreases. And when fear of the Hun, or of personal death or injury, or when loss of property, hunger, sex desire, mob activity or race hatred rise above intellectual ideals, is not morale gone? Without morale an army is ready for defeat and rout. Without morale, an individual is ready to develop a psychoneurosis.

Abstracts from Current Literature

ABSTRACTS OF SOME RECENT WORK ON THE NEUROGLIA BY THE SPANISH SCHOOL

(Continued from page 552)

HISTOLOGICAL AND HISTOPATHOLOGICAL INVESTIGATIONS ON THE PINEAL GLAND IN MAN.

N. Achucarro and J. M. Sacristan, Trab. d. lab. de invest. biol. Univ. de Madrid **10**:185, 1912.

The authors give a summary of the bibliography, treating especially the work of Dimitrowa (**2**:3, 1905, of Studuicka [Jena] Cutore, Bizzozero, Le Neuraxe), Meynert, Hagemann, Henle, Cionini, Weigert, Cajal, Krabbe and others.

The authors use these stains: toluidin blue, hematoxylin-eosin, Van Giesson, Alzheimer, Mallory, Weigert, Bielschowsky, Heidenhain and Cajal.

1. Connective tissue passes from the pia mater along the vessels, forming an adventitial sheath to vessels thus furnishing adventitial lymph spaces as in the vessels of the brain. This space is more dilated in pineal of regressive character. In such cases the vessels are tortuous and form pockets as in atrophic conditions of the brain. The connective tissue separates the vessels from the parenchyma and also forms strands dividing the organ into lobules and lobes of irregular size. The lobules are not always well limited.

Four types of connective tissue organization are seen: (*a*) Trabecula not forming a network, but passing simply from one point of connective tissue sheath to another; (*b*) fine network of fibers; (*c*) coarse network of trabeculae complicated in arrangement; and (*d*) many serpentine filaments, tendril like, recalling the appearance of elastic fibers (not seen in ox or sheep). These fibers resemble ovals and knots described by Cerletti in senile atrophy and are observed most in pineals which show atrophy or other marked regressive phenomena.

In some parts of the gland there is no appearance of lobulation, the connective tissue forming a thick network. In the perivascular spaces of the pineal of both man and beast mast cells are seen. Plasma cells are noticed in man and in ox, sometimes in groups, but no lymphocytes are seen with them. Granular cells are seen in the pineal of man, containing yellowish or green pigment, in cases where other marked regressive phenomena are present, tortuous vessels, dilated lymphocytes, retraction of parenchyma, etc. Pineal "sand" is seen in perivascular spaces.

The cells of the lobules are mostly nerve cells with vesicular nuclei, the membranes of which are folded and the nuclei contain frequently the so-called "Dimitrowa balls" which, in the authors' estimation, are due to foldings and inclusions of the nuclear membrane (many illustrations are shown elucidating the process). These inclusions are not always spherical, but frequently oval, oblong, etc. Bonfiglio has described like cells in severe degenerative brain disease. These cells with inclusions are much more plentiful in old age and in regressive conditions in the pineal such as connective tissue proliferation, etc. The authors, therefore, consider these "balls of Dimitrowa" as products of regression of the nuclei and not evidences of glandular secretion.

Glia cells are found in the lobules and a rich plexus of neuroglial fibers. A few medullated nerve fibers are seen, but many more nonmedullated fibers. In the periphery of the lobules and in the perivascular spaces cells are seen with many prolongations terminating in buds around the vessels. These cells contain fibrils in their protoplasm and appear to be sympathetic nerve cells. The authors' later researches show that these "end feet" and many branched fibers are proliferative changes in a cell reacting to degenerative changes in the neighborhood as demonstrated by many granular cells, etc. Connective tissue cells are seen sparsely in the lobules near the trabeculae, etc.

CONTRIBUTION TO THE KNOWLEDGE OF THE NEUROGLIA OF THE HUMAN CEREBRUM. S. Ramon y Cajal, Trab. d. lab. de invest. biol. Univ. de Madrid **11**:255, 1914.

The author concludes that:

1. Fibrous astrocytes and protoplasmatic astrocytes both originate in epithelial cells or spongioblasts of the medullary tube. They represent the results of the adaptation of an identical primitive type to two different environments. Therefore, the mesodermic origin of the glia claimed by certain authors is untenable.

2. During embryonic life and after birth the dislocated epithelial cell and astrocyte are capable of proliferation in normal circumstances.

3. The protoplasmatic astrocyte has a structure recalling that of glandular cells: in the midst of a dense spongioplasm gliosomes or differentiated granules lie. Their number, size and staining reactions vary according to functional states.

4. The interstitial net in the gray matter of cortex described by many authors is not neuroglial in nature. The ramifications of the glia form a very complex plexus.

5. Every astrocyte of both gray and white substance is provided with a suction tube or perivascular foot. This is delicate in the protoplasmatic and robust in the fibrous astrocyte.

6. All astrocytes of both man and beasts, without exception, possess a centrosome. In young animals the endocellular apparatus of Golgi is also seen. Both are seen in the area of maximum protoplasm.

7. A small adendritic body exists in the nervous centers, probably mesodermal in origin, certainly neither belonging to nervous nor to glia tissue.

8. Aside from other possible varieties of the above cell, two types are seen in all likelihood of different activities—the perineuronal or satellite type, probably the homologue of the subcapsular elements of the ganglions, and the apolar cell of the white substance, probably corresponding to the Schwann's cell.

9. The adendritic cells of the white substance may comprehend diverse physiologic categories of the mesodermal elements. Among the adendritic cells remnants of astrocytes in involution probably are present.

10. During the epoch of evolution astrocytes are capable of phenomena of migration and expansional transformation which are similar to the ameboid movements of leukocytes. In this way vascular feet are formed which sometimes are the result of a new expansion, at others of the displacement and increase in a radial or primordial prolongation.

11. The Ranvier-Weigert fibers result from the intraprotoplasmatic differentiation of the astrocytes. In no case do the fibers completely free themselves from the glial protoplasm.

12. The human cortex differs from that of animals, not only in the enormous quantity of the glandular type of cells which it contains, but also in their small size, the great richness of the interstitial glial plexus and the absence of any tendency in the glandular type of glia to differentiate intraprotoplasmatic fibers in normal conditions.

CONTRIBUTION TO THE STUDY OF THE NEUROGLIA OF THE CEREBELLUM. J. RAMON Y FANANAS, Trab. d. lab. de invest. biol. Univ. de Madrid **14**:163, 1916.

The author uses original Cajal fixation, sections from 20 to 25 microns placed from four to six hours at 18 to 20 C. in gold-sublimate bath. Achucarro's tannin silver shows the molecular layer, but not others.

The molecular layer of the human cerebellum shows protoplasmatic astrocytes of four types: (*a*) mono-radiated, sometimes twin; (*b*) bifurcated villous type, mostly near the Purkinje cells; (*c*) protoplasmatic type, many ramifications, generally in about the middle of the molecular layer; (*d*) perivascular astrocyte—rather rare; it has protoplasm concentrated in upper part of the nucleus, is vacuolated, has one large branch ascending giving off many fine short branches laterally and one short thick branch which lies along a capillary. Glial feet are seen only in class *d*. Autonomous and polarized cells show ependymal origin. In the rabbit there are many glial feet. Epithelial or forked cells or Bergmann's fibers are seen in all animals and man, starting in the Purkinje layer. Their nuclei are pale purple with black granules. The cell is vacuolated and has two or three long branches with more or less protoplasm. Dislocated epithelia cells are found in all parts of the molecular layer. Satellite cells of the molecular layer, both astrocyte and adendritic (nonglial) are found. The glia of the granular layer consists mostly of transitional cells, one portion of the branches is protoplasmatic, other portions are fibrous. Some fibrous astrocytes are found near the Purkinje cells.

The neuroglia of the white substance is fibrous in type, somewhat larger than in other portions of the brain. Many adendritic cells are seen.

Conclusions: 1. Molecular layers contain autonomous astrocytes, Bergmann's fibers and prolongations from cells in the granular layer. Most astrocytes of class *d* insert at least one expansion on a vessel; they contain many gliosomes. 2. About the Purkinje cells both fibrous and protoplasmatic astrocytes are found; also certain cells applied to the neuronic dendrites which may serve as insulators. 3. Adendritic cells are frequent in all parts. 4. The protoplasmatic cells contain gliosomes.

VARIATIONS IN THE HISTOPATHOLOGY OF THE NEUROGLIA IN CEREBRAL SOFTENING. P. DEL RIO HORTEGA, Trab. d. lab. de invest. biol. Univ. de Madrid **14**:1, 1916.

The author treats in great detail the regenerative types of glia seen in and about a softening: hyperplasia, hypertrophy of cells and fibers, "monster" forms of cells (certain types at first progressive later become regressive), vacuolar degeneration of dendrite masses: segmentation of the prolongations in the form of rings, rackets, clubs, etc.

Regressive phenomena coincide in time with progressive. Noted among the regressive were klasmatodendrosis beginning at the periphery and extending to body of cell and the formation of "füll Körperchen."

In studying granular cells the author has never seen anything that would lead him to infer that the glia took any part in their formation; on the contrary, he has seen many transitional forms between the adendrites or third element and the granular cell. He considers the adendritic cell the predominating type of granular cell and recognizes in addition only cells from the blood vessel in the same capacity. He distinguishes the adendritic cell and its transitional forms up to the true granular cell by the richness of its nucleus in chromatin—the neuroglial cell nucleus is poor in chromatin.

The author considers the great difference of opinion of different observers in regard to the granular cell due to the nonrecognition of the adendritic cell as a nonglial body.

ALTERATIONS OF THE CENTRAL NERVOUS SYSTEM IN A CASE OF PARALYTIC FORM OF DISTEMPER IN A DOG.

PAUL RIO HORTEGA, Trab. d. lab. de invest. biol. Univ. de Madrid **12**: 97, 1915.

The author reviews all the work done on the subject up to date and then gives the results of his case studied by the methods of Cajal and Bielschowsky for neurofibrils and axis cylinders, by Nissl's toluidin blue and Mann's stain for the chromatic portion of the nerve elements, by Weigert's and Mallory's glia stains and Achucarro's glial and connective tissue stains, by Unna-Pappenheim's stain, toluidin blue, and iron-hematoxylin for inflammatory elements, and by Herxheimer's stain for disintegration products.

His conclusions are:

1. In distemper of the paralytic form an inflammatory process exists in the central nervous system that may be designated as a diffuse meningoencephalitis.

2. This process is characterized by foci and plaques of cellular infiltration specially localized in the gray matter of the cerebrum in connection with meningeal inflammation and in the white matter of the cerebellum.

3. The lesions of the cerebellum precede those of the cerebrum and in the latter superficial lesions precede the deeper.

4. Vascular dilatation exists in early foci of infiltration. Dilatation of the adventitial lymph spaces, increase in the adventitial elements and large accumulations of small lymphocytes and plasma cells and some polynuclear leukocytes are always present in the foci.

5. Newly formed connective tissue is present in the oldest foci and in the diffusely infiltrated areas of the cerebellum. This connective tissue invades the nervous tissue and forms a complicated network in whose meshes various classes of cells are lodged (lymphocytes, plasma cells, neuroglia and ameboid).

6. The nervous elements undergo regressive phenomena consecutive to the presence of inflammation and the presence of the causal agent of the disease.

7. The above-mentioned regressive phenomena consist, in the cerebrum, of atrophy and marginal vacuolization of the cortical cells, retraction and pyknosis of the nuclei, hypertrophy and multiplication of the nucleoli and dilatation of the medullated fibers. In the cerebellum, like degenerations are seen in the

Purkinje's cells, formation of knobs and club-shaped bodies by retraction of the axons, retraction and enlargement of the pericellular nets and vacuolization and breaking of the fibers of the white substance.

8. Parallel to the cellular destruction in the cortex there is new formation of glial elements specially of the glial fibers, with abundance of astrocytes and some rod cells.

9. Preameboid elements are plentiful in the cerebellum as are also phagocytic ameboids acting on the destroyed axons.

10. In some ganglion cells in the base of the brain and in the peduncles very small intraprotoplasmatic and intranuclear bodies are seen which show a similarity to the smallest forms of protozoa studied by Noguchi in rabies and to the "Staupe Körperchen" described in distemper by Lentz, Standfuss and Sinigaglia.

11. Although the inflammatory lesions in distemper coincide with those of rabies, the reactive phenomena of the nerve cells to the respective viruses is not identical.

12. In relation to general paralysis of the insane, a great similarity of the inflammatory process and of some secondary results, such as the connective tissue sclerosis, is seen.

DENDRITIC RAMIFICATIONS, NEWLY FORMED, IN NERVE CELLS, AND ALTERATIONS IN THE NEUROGLIA IN THE SENILE DOG. Gonsalo R. Lafora, Trab. d. lab. de invest. biol. Univ. de Madrid **12**:39, 1914

The pyramidal cells in Ammon's horn of the senile dog present numerous lateral ramifications of their dendrites, numbering twenty or more, of greater or lesser length, but in general ending not far from their point of origin. These ramifications are not seen on all the dendrites, but generally on the principal dendrite shortly after leaving the body of the cell.

This alteration is seen in small foci, yellowish in color (in silver preparations). Under high power an area is seen without intercellular fibrils, but filled with a yellowish homogeneous substance in the midst of which are seen two or three neuroglial nuclei and the dendrite from which the newly formed ramifications originate. As a rule the new ramifications do not surpass the yellowish zone. The ramifications show numerous neurofibrils by Bielschowsky's method. The ramifications are tortuous, show irregular enlargements and often terminate in bud form.

Nuclear stains (toluidin blue, etc.) do not stain the homogeneous yellowish substance, and the glia around it shows no proliferative reaction. The author considers the dendritic new-formations as a reactive or regenerative reaction similar to the formation of senile plaques. The cause of this reaction is excretion from the nerve cell.

Senile plaques in the vicinity of blood vessels are frequent and explained by the action of the lymph flow to the vessels carrying the irritating substance with it.

The irritating substance is shown to be of neuronic origin because plaques are encountered only in the cellular portions of cerebrum or cerebellum, in other terms, only where nerve cells exist, and because plaques are never found in other portions of the nervous system to which their formation could be ascribed (neuroglia, axis cylinders, etc.).

Certain alterations of the dendrites somewhat similar to those described by the author have been seen in the spinal ganglions in senility (Cajal, Trabagos **4**: 1906), and in the dendrites of the Purkinje's cells after traumatism (Cajal, Trabagos **9**:1, 1911) and in the Purkinje's cells in general paralysis (Ruiz de Arcaute, Bull. de la Soc. Española de Biologia [Jan.] 1912).

The neuroglia in senile degeneration in the dog shows marked changes seen in the hypertrophy of the glia cells and the thickening of the cellular processes.

SOME OBSERVATIONS ON THE HISTOLOGY OF THE HYPOPHYSIS (HUMAN). F. TELLO, Trab. d. lab. de invest. biol. Univ. de Madrid **10**:145, 1912.

A. Anterior Lobe.—The epithelial cells of the anterior lobe contain a network of fibers stained only by methods that stain in the same manner the mitochondrias and the ergastoplasm (Bonin, Da Costa). These fibrils are not stainable by any method that stains the neurofibrils. The theory is that they represent various stages of function. Nerve cells with one, two or three dendrites and large nuclei are seen.

A variable number of nerve fibers are seen, sometimes many, sometimes few, but always in determined regions, especially close to the upper surface, to the posterior lobe and to the anterior pole. They pass along the capsule, pierce it and enter the glandular substance either passing along the connective tissue or going freely among the cells and ending usually in balls or globular distentions. They frequently throw off branches as they go and sometimes are lost by the fineness of their branching. Some fibers approach the rare nerve cells, above spoken of, and are probably the sympathetic fibers from the carotid plexus described by Luschke.

B. Posterior Lobe.—The author has never been able to find nerve cells by any methods or at any age from infancy to senility. Nerve fibers in great fascicles enter by the peduncle, separate from time to time to give passage to tortuous blood vessels, and these fibers, the vessels and glial fibers fill the whole space of the peduncle (the tortuous vessels suggest senile atrophy). On reaching the lobe these fascicles separate and the separate nerves divide forming one of the richest nerve plexes in the nervous system. All these fibers are nonmedullated. These fibers are so complicated that the author has never been able to trace their endings.

Mesodermal Tissue and Neuroglia: There are two types of cells (1) glia cells: star shaped with large nucleus which is spherical, elliptical or ovoid; abundant protoplasm prolonged into abundant ramifications; (2) the second cells are large and fusiform with a rod-shaped nucleus and protoplasm accumulated at the poles—rod cells and fibroblasts. The amount of connective tissue around the vessels and between them is very considerable.

C. Intermediate Lobe.—In infancy and childhood this is usually nothing more than a cleft lined by columnar epithelia, separating the two lobules. In later life this cleft forms many cysts. These vesicles vary in lining, but the cells are mostly flattened and polymorphous, and in several layers. Colloid material and sometimes granular is seen in the vesicles. Some cells appear ameboid and are phagocytic.

Some bipolar cells are seen, probably sensory cells. There are a moderate number of nerve fibers; they form ramifications around certain ovoid epithelial cells.

D. Pathologic Modifications of Hypophysis.—Fibers are seen in degeneration; fragmentation of a fiber takes place into a series of spheres which gradually lose all structure and become pale and finally almost invisible. All stages are seen, some showing axon and retracted fibrils, etc. These degenerated fibers are seen at all ages and in all stages of health, but in the infant and healthy adults they are scattered and distinct whereas in cases of softenings, hemorrhage, neoplasms, etc., they form foci. In old age basophil cells invade the neurohypophysis and act as neoplasm, crowding and destroying the fibers.

Regeneration of the nerve fibers takes place to a limited degree, e. g., the fibers sprout, terminal arborizations take place in unaccustomed places, masses of fibers occur, such as are seen in newly-forming nerves, the neurofibrils are separated and individualized, etc.

Granular Cells Present in Pathologic Conditions: Two varieties at least are to be noted — the ordinary active phagocyte of Merzbacher, round, ovoid or slightly elongated with coarse spherical granules and cells of the spindle-shaped variety, the nuclei of which become so obscured by the large irregular granules that they have the appearance of cylinders not resembling any cell form. Many of these cells are parallel with the vessels or with connective tissue lines. Such cells are seen isolated in youth, but increase progressively with age, just as degenerative phenomena take place in the nerve cells and fibers, and in old age these granular cells are plentiful and grouped into foci. In the case of invasion of the posterior lobe by the basophilic cells, the granular cells are in great numbers, and are seen grouped around the foci of invasion. The same thing is observed in hemorrhages, softenings, etc. The author saw large quantities of them in an idiot of 25 years and in a puerperal patient.

The author considers the second cells to be the passive variety of phagocytes described by Merzbacher, originating in the glia cells and particularly engaged in taking up the products of nerve fiber degeneration.

GURD, Ann Arbor, Mich.

LA FONCTION PSYCHO-MOTRICE D'INHIBITION ETUDIEE DANS UN CAS DE CHOREE DE HUNTINGTON. ESSAI D'APPLICATION DES TECHNIQUES DE LA PSYCHOLOGIE EXPERIMENTALE A LA NEURO-PSYCHIATRIE. RAOUL MOURGUE (Villejuif, Seine), Schweiz. Arch. f. Neurol. u. Psychiat. **5**:70, 1919.

Considering the number of monographs that have been devoted to the study of divers psychoses one is struck by the paucity of investigations on the psychic aspect of chronic chorea. The discovery of definite encephalic lesions in chronic chorea by Pierre Marie and Lhermitte places this malady definitely among the organic diseases of the brain.

The author had occasion to study a patient with chronic chorea of three years' standing, who exhibited the usual neurologic manifestations seen in this disease. He had in addition a right supranuclear facial paralysis, the significance of which is not explained. It might be said that shortly before his admission to the hospital he was in an automobile accident in which he lost consciousness. His face was mask-like even during violent outbursts of temper, which he frequently exhibited; the only complaint in addition to the choreoathetotic movements was a "thickening" of the tongue. The oculocardiac reflex was absent.

The principal mental features consisted in marked difficulty in calculation, inability to describe accurately objects which he had seen, particularly if this involved spatial localization, and a mild psychosis with a persecutory trend. The writer insists that in this case, at least, the dementia was more apparent than real, since the principal difficulty was in recalling, rather than in a complete loss of facts; the inability of the patient to put these into service betrayed an interference with so-called intellectual autoconduction. If one insisted or tried to aid mental imagery, for example, by asking the patient to make a diagram of the Place de la Concorde, he could do this with remarkable accuracy. Another characteristic trait was the irritability, which was dependent on a transformation of character, also an expression of this loss of autoconduction.

The author adheres to the point of view which ascribes psychophysiology to the functions of the central nervous system, in contradistinction to the theory of W. James. Pagano has shown that chemical excitation of the anterior and middle thirds of the head of the caudate nucleus, particularly the internal portion of it, will in dogs provoke emotional phenomena which present all the characteristics of fear. Excitation of the posterior third of the same nucleus gives rise to an ensemble of phenomena attributable to anger. He refers to the conception which looks on the central gray nuclei as an apparatus for vasomotor control, although this has not been definitely demonstrated to be the case in man. He quotes Brouwer to the effect that the caudate nucleus subserves the functions of the sympathetic system, particularly in its action on the smooth muscles, while the lenticular nucleus is related to reflex movements of the striated muscles.

Chronic chorea involves, with a remarkable predilection, the frontorolandic area and the corpus striatum. Lesions of the fibers are of particular importance; the tangential, the supraradial and the intraradial fibers showing degeneration. In the frontal area there is an absolute disappearance of tangential fibers; in the rolandic area this change is marked; while in the parietal and occipital areas the fibers appear normal. The subcortical white matter, while it appears normal superficially, is in reality somewhat degenerated. Normally the cerebral cortex inhibits and controls the subcortical motor centers. Lesions, therefore, that involve the corticostriate and corticothalamic fibers permit the subcortical centers to rise to uncontrolled activity. The writer again refers to the experiments of Pagano, and feels that this explains in a satisfactory manner the transformation of character seen in chronic chorea. The irritability is more or less automatic and is endogenous in origin. The mechanism of attention is likewise essentially motor, the degree of concentration being inversely proportional to the quantity of active external diffusion by muscular activity.

The writer reports a number of experiments undertaken with the purpose of showing the inhibitory effect of the corticothalamic system. In the first of these he makes a tracing, with the aid of Marey tambours and a revolving drum, of the horizontal, vertical and sagittal movements of the arms. The patient was commanded to inhibit the movements as much as possible at certain intervals. Absolutely no evidence of inhibition was noted. The writer concludes that the incoordination of movement is the result of lesions related to the corpus neostriatum—that is, the caudate nucleus and the putamen, the functions of which are supposed to be inhibiting and coordinating.

A second experiment as made by recording the patellar reflexes, the patient being asked to resist the movement. Here, too, there was no evidence that

the patient was able to comply. The same thing occurred in commanding the patient to inhibit the movements of the eyelids. Tracings were also made of the respiratory movements, but gave no positive proof that the patient was able to arrest these movements on command. He then applied the inhibition test of Patrizi, in which the plethysmograph is applied to the hand and changes in volume recorded. The patient is then told to remain as quiet as possible, while a loud noise is made by striking a violent blow on a plate; the test is then repeated, but without warning the patient of the coming noise. It was interesting to note that the patient in both of these tests exhibited absolutely no evidence of vasomotor excitation, which is so striking in normal persons. The same thing occurred when the patient was reminded of the loss of 105 francs, which to him was a very serious matter. Even reference to the fact that he was being pursued produced no response.

Even with this state of continuous and profound distraction, it could not be denied that he was much more irritable than before the onset of his illness. The author believes that the method he employed in obtaining the reaction time is preferable in this type of case to that of association, since the interpretation of the results gained by the latter is infinitely more complex and does not lend itself so readily to numerical evaluation.

WOLTMAN, Rochester, Minn.

POLIOMYELITIS. A STUDY OF THE 1916 PHILADELPHIA EPIDEMIC WITH A REPORT OF 717 CASES. T. H. WEISENBURG, Tr. Amer. Neurol. Assn., 1918.

Weisenburg has given the results of a thorough analysis and clinical study of 717 cases of poliomyelitis which came under his observation in Philadelphia during the epidemic of 1916. Of these, 388 were in males and 329 in females. The ages ranged between less than 6 months to over 30 years, 63 per cent. occurring between the ages of 6 months and 3 years. The total mortality was 22.8 per cent.

Preparalytic symptoms are considered under the heads of fever, respiratory and pulse rates, gastro-intestinal symptoms, nervous symptoms, respiratory symptoms, pain, retention of urine, sweating, chills, nosebleed and objective preparalytic symptoms. Objectively, the appearance in this stage was similar to that of meningitis, with stiffness of the head and neck, often with retraction of the head, general hyperesthesia, increased activity of the deep reflexes and positive Kernig sign. Fever was invariably present and of a very definite type. It was usually moderate, 100 to 103 degrees, with but slight diurnal variation. The duration was rarely over six or seven days unless some complication supervened. The respiratory and pulse rates paralleled the temperature curve. Nausea and vomiting were common; irritability and twitching occasional; delirium, stupor and convulsions rare.

The onset of the paralysis was noted quite uniformly on the second or third day, the paralysis rapidly extending to its most severe degree. Relapses were never observed. "A paralysis which tends toward increased destruction of function after improvement has been instituted should cause grave doubts as to the etiology being infantile paralysis." Pain in the head, neck and back was almost invariably present in the patients who were old enough to describe their sensations. The pain is not of neuritic, but of meningeal origin and disappears as the signs of meningeal irritation subside or when the intracranial pressure is relieved by lumbar puncture. The spinal fluid was found uni-

versally under increased pressure. The appearance was clear and colorless or with but faint opalescence. After standing, 10 per cent. of the fluids presented a small fibrin coagulum. A moderate lymphocytosis was found in the preparalytic stage and continued for about two weeks after the onset of the paralysis. An increase of globulin was found in 32 per cent. A colloidal gold reaction of the meningitic type was present in from 40 to 50 per cent. of the fluids during the acute stages. While "a clear or slightly opalescent fluid, poor in fibrin, reducing Fehling's solution and containing an increased number of cells chiefly of the mononuclear variety, are the most constant findings," there are no definite diagnostic features in the spinal fluid.

The extent of the paralysis could be determined a few days after the paralysis became manifest and was usually severe. The types of paralysis are divided into the following groups: (1) the spinal form; (2) the form resembling Landry's paralysis; (3) the pontine bulbar: (a) bulbar, (b) pontine, (c) pontine bulbar, (d) pontine spinal, (e) bulbar spinal, (f) pontine bulbar spinal; (4) encephalitic; (5) cerebellar; (6) meningitic; (7) abortive. In regard to the recession of paralysis, the cranial nerve palsies and manifestations of cerebellar involvement usually cleared up promptly. "Distal muscles of any particular extremity cleared up more rapidly than the proximal muscles. . . . The rate of improvement in any given extremity may be the same, but there is a more rapid improvement in the upper extremities when contrasted with the lower extremities."

Careful consideration of the question of contagion shows little evidence that the disease is personally contagious.

Various methods of treatment were used including urotropin by mouth, normal serum intraspinally and intravenously, mercuric chlorid solution intraspinally and arsenobenzol intravenously; also immune serum intraspinally and epinephrin intraspinally as advocated by Meltzer, but with little conviction as to the value of any of these agents.

This short, concise article is the result of a careful study of a large number of cases, and forms a valuable addition to our clinical knowledge of this disease.

Howe, New York.

WAR NEURASTHENIA, ACUTE AND CHRONIC. D. W. Carmalt Jones, F.R.C.P., Brain **42**:171 (Oct.) 1919.

In a careful study of 1,300 war neurasthenics, Jones emphasizes the physical aspects of the problem. Of the entire group, 612 cases were acute, in the sense that the neurosis was abruptly precipitated; 334 chronic, being usually the outgrowth of prolonged and insidious strain and in 354 the condition was complicated by organic disease. War neurasthenia is defined as: "A state of fatigue of the central nervous system, without organic change, manifested by asthenia, loss of physical and emotional control and disturbances of visceral functions." Briefly, the cause is a question of physical and emotional fatigue. As precipitating factors, personal "nerve" and resisting power are especially taken into account. The former concerns the soldier's record in civil life, information as to whether he has ever suffered from a "nervous" breakdown being of particular importance. As might be expected, a "bad nerve" record was fairly frequent. The author also found that the resisting power is less affected by the circumstances that apparently give rise to the neurosis, than it is by the personal qualities inherent in the individual.

Further, while this may be a considerable factor in a chronic case, it is of far less significance in an acute one. The symptomatic findings may be expressed in the following statement: "The commonest symptoms are those of common sensation in excess, disturbance of sleep, and disturbance of coordination; pure motor symptoms, with the exception of asthenia, are rare, visceral disturbances and also psychical symptoms are common though not invariable, and disturbances of the special senses are infrequent." Headache was present in 736 patients, disturbed sleep in 515, incoordination in 374, pain, other than headaches in 303, asthenia in 266, alimentary symptoms in 251, cardiovascular symptoms in 237, urinary symptoms in 204, psychic symptoms, aural symptoms in 191, respiratory symptoms in 104, ocular symptoms in 92, mutism in 85, paresthesia in 78, paresis in 41 and anesthesia in 37. The prognosis was usually good. Fifty per cent. of the patients with acute cases were returned to field duty and only 16 per cent. were sent to base hospitals. However, only 20 per cent. of the "chronic" neurasthenics were able to rejoin their former commands. The treatment was rest, graduated exercise, maintenance of discipline and mental occupation.

The author's recommendation that these patients be treated at a point far remote from the firing line must be seriously questioned. It is, of course, obvious that frequent artillery fire and bombing raids are not in themselves conducive to recovery from war neuroses. However, treatment near the front has certain advantages which outweigh its contraindications. The neurosis is less apt to become fixed when the patient is kept in the midst of stimulating military activity and the necessity of quickly returning to duty is constantly and strikingly suggested to him. In quiet areas, away from the "noise of guns," the urge is far less great and the stage of symptom-fixation is correspondingly easy (reviewer's opinion). I believe that the experience of divisional neuropsychiatrists who were with the A. E. F. will bear out these statements.

STRECKER, Philadelphia.

A TRIPLE OBSERVATION OF FAMILIAL PSYCHOSES IN A FAMILY OF NEGROES. E. TERRIEN and R. SAQUET, Progrès méd. **35**:72 (Feb. 14) 1920.

The authors report a negro family in which the father, a Senegal negro, and his two mulatto daughters, aged 18 and 19, developed psychoses. The former, a case of paranoia, was dominated by a paranoid idea, persistent during twenty years, on an invariable theme (his sonship to an Ethiopian king) without any intellectual enfeeblement. His original character shows inherent traits of a paranoid constitution—excessive egoism and pride. The daughters had dementia praecox with marked paranoid coloring. In them, intellectual impairment was progressive. The authors debate whether they were dealing with true familial mental disease as studied by Féré, Trénel, Fougue, Apert and Demay and conclude that they were.

These considerations are outlined: Demay has written that there is no parallelism between familial mental illness and familial affections of the central nervous system, such as Friedreich's ataxia, Landouzy-Déjerine myopathies or Thomsen's disease, because familial mental disease is not *sui generis*. Rather, the expression "familial psychosis" simply characterizes a resemblance which sometimes exists between psychoses in members of the same family. In the family cited, the paranoid feature is the common characteristic and alone justifies the term "familial" in their opinion.

Apert has pointed out that a psychosis to be classed familial must be outside of an exterior pathology (intoxication, infection, organic cerebral affections). Dumas does not believe that persuasion or example account for familial psychoses, though "persuasion may provoke the hallucinatory modifications." The authors consider that in their cases persuasion or example was not a factor as the death of the father occurred prior to the onset of symptoms in the daughter. They, perhaps, at this point, stress insufficiently the example set by the father in the preceding years—those during actual adolescence.

They elicit the law of progressive degeneration of Morel to explain one feature—namely, the dementia of the second generation in sharp contrast to the preservation of intellect in the father. They think that true paranoia cannot reproduce itself in a successive generation. Rather, it is a grain which begets in the following generation a thing more degenerative and destructive —paranoid dementia praecox.

DAVIS, New York.

A METHOD OF TREATMENT FOR NEUROSYPHILIS. J. A. KOLMER, J. A. M. A. **74**:794 (March 20) 1920.

Kolmer's technic seeks to avoid the disadvantage of the Swift-Ellis treatment by providing for the introduction of a larger amount of arsphenamin into the spinal canal. Advantage is taken of preliminary spinal puncture, which is said to increase vascularity of the meninges and favor absorption by the drug.

A "course" of treatment covers four weeks, and comprises four combined intravenous and subdural injections of arsphenamin at weekly intervals. The first injection is preceded by a week of daily mercurial inunctions and potassium iodid by mouth. The inunctions and the iodid are kept up throughout the course.

The technic of injection is:

Six-tenths gram of arsphenamin is dissolved in normal salt solution, neutralized and the volume brought up to 200 c.c.; 1 c.c. of this is removed for further use. The remainder is injected into a vein of the patient and immediately thereafter 25 c.c. of the blood are withdrawn from another vein. This is mixed with 4 c.c. of 10 per cent. sodium citrate solution and centrifuged. About 10 c.c. of the resulting blood plasma is then mixed with 0.1 c.c. of the arsphenamin solution (dosage 0.0003 gram). As high as 0.3 c.c. of arsphenamin solution may be used in subsequent treatment. The arsphenamized serum is heated at 56 C. for thirty minutes.

Spinal puncture is done at once with a small (1 mm.) needle to ensure a slow fall in pressure. About 30 c.c. are withdrawn and the serum then slowly injected by gravity or by syringe.

Blood withdrawn immediately after injection is said to contain more arsphenamin than that withdrawn one hour after injection. The total time of the operative procedure is about two hours. In case of reaction to arsphenamin the intraspinal injection may be reserved for twenty-four hours.

HAINES, Philadelphia.

CEREBROSPINAL MENINGITIS. M. Aynaud, Progrès méd. **35**:45 (Jan. 31) 1920.

Regarding the four known types of meningococci, the author comments on the recent increased frequency of types B and C which now furnish more than one half of the cases of cerebrospinal meningitis. Type D remains rare clinically. Types A, B, C and D have the same cultural characteristics, including those of fermentation, and differ only according to their reactions to serums, where their agglutinations are specific. Therefore, the author insists that serum therapy should be based on type and that each case should receive the expert bacteriologic care necessary to establish the type before the serum therapy is begun. He has found only the most ultra aseptic technic satisfactory, and warns against any method other than drawing the spinal fluid into an autoclaved syringe which is then emptied into a flamed sterile tube.

The second portion of the article discusses the complication of pyocephaly and ventriculitis. In contrast to the fever, Kernig's sign and stiffness of the neck which are signs of meningeal infection, psychic, trophic and sphincteric troubles and violent headache are the symptoms of cerebral compression. They establish themselves as the ventriculitis develops. When the course indicates this complication, treatment should be by the ventricular route as well as by the lumbar. "If at the end of five or six days of correct and intensive treatment the fever and other symptoms persist along with continued purulence of the fluid, and if, in addition, a careful examination shows no septicemia or extrameningeal localization of the meningococci, I consider it proper to incriminate the ventricles and to have recourse to ventricular puncture." By the lumbar route, the author injects from 30 to 40 c.c. of serum after having withdrawn the same amount of fluid, but by the ventricular route he withdraws only from 15 to 20 c.c. and injects in serum an amount somewhat smaller. When ventriculitis has developed and the fluid remains purulent, he gives both a lumbar and ventricular treatment each day.

A third portion of the paper deals with anaphylactic sensibility, and procedures of desensitization are given. These are not new. In order to gain a perfect security against anaphylaxis, even though the patient is supposedly desensitized, he inaugurates intraspinal serum treatment as follows: He places 0.5 c.c. of the serum in a syringe and then aspirates 10 c.c. of the spinal fluid into it. This is allowed to mix and the mixture is immediately injected. Then with the needle remaining in place, he waits three or four minutes, after which he withdraws the customary 30 or 40 c.c. of fluid. After this, he finds the injection of the serum invariably free of untoward symptoms.

Davis, New York.

A PROPOS DU SONDAGE NASAL DES ALIENES. Will Boven (medécin à Cery), Schweiz. Arch. f. Neurol. u. Psychiat. **5**:99, 1919.

Boven lays stress on the importance of persuading patients to eat in a normal manner, and employs tube feeding only as a dernier ressort. As might be expected, patients, particularly those having mental disorders, react very differently, depending on the type of psychosis. Tube feeding by mouth usually is not only very difficult, but is also exceedingly uncomfortable, as far as the patient is concerned. Boven, therefore, uses the nasal route almost entirely. By experimenting on himself, and in response to inquiries among his patients, he concluded that the greatest pain in nasal feeding was the result of pressure

on the inferior turbinate. This leads to pain referred to the superior molars, the nose and the temple of the homolateral side, the reflex path being the sphenopalatine ganglion and branches of the trifacial nerve. When a large tube is used, this is particularly severe. The pressure of a large tube in the vicinity of the larynx can also separate the vocal cords so that any regurgitated liquid readily enters the glottis. He has adopted the following procedure: The left nostril is sprayed with a 20 per cent. solution of cocain, after which a tube having a caliber of 5 to 8 mm. is inserted. At the moment the patient swallows, during which time the glottis is closed, the tube is rapidly passed into the esophagus and down to the stomach. Should the trachea have been entered, this is at once shown by a violent paroxysm of coughing which occurs after a few drops of water enter the lungs and through the inability of the patient to make an audible sound. In a selected number of mental cases he gradually discards the use of cocain and resorts to larger tubes, thus producing a gradual increase in the amount of pain, which not infrequently has the psychologic result of encouraging the patient to feed himself.

WOLTMAN, Rochester, Minn.

LES TROUBLES OCULAIRES AU COURS DE LA THROMBOSE JUGULAIRE DU CARDIAQUE. V. CORDIER and P. ROLLET, Progrès méd. **35**:108 (March 6) 1920.

A soldier with a history of rheumatic fever at 16 and again at 23 developed temporary cardiac symptoms at the age of 29. These responded to treatment but a year later (February, 1917) decompensation recurred and progressed to a fatal termination in four months. Ocular signs which are not easily explained even with the aid of careful necropsy search are the subject of the writers' interest.

The case was one of mitral stenosis and insufficiency with pericarditis. A thrombophlebitis of the great veins draining the right upper extremity developed and there were multiple and repeated infarctions of the lungs. For forty-eight hours before the patient died there was unilateral exophthalmus (right) with diplopia and also abolition of the light reflex in the right eye. The diplopia was not accompanied by ocular paralyses nor the light reflex loss by loss of the reflex on accommodation. There was no pain on pressure on the eyeballs, no signs of meningitis, no vertigo, no troubles of ideation or of language, and audition was normal. The eyelids were not edematous and showed no painful or indurated bands. The orbital and peri-orbital regions were free from swelling. The conjunctivae showed no injections. There was slight congestion of the fundus though the large vessels were not turgescent and there was no retinitis or hemorrhage. Necropsy showed a thrombophlebitis of the right innominate vein beginning at the superior vena cava. It extended upward 3 cm. in the internal jugular and 6 cm. in the external jugular and extended laterally a distance of 2 cm. in the subclavian. Careful search was made in the cranium. There was a slight degree of meningeal congestion. There were no retro-ocular effusion, no tumor, abscess or aneurysm. The arteries were normal and nothing abnormal was found in the veins or sinuses. The cavernous sinus was especially explored and found clear as were the tributaries, the ophthalmic veins. All the bony sinuses were free of infection.

The few ocular signs suggesting a cavernous sinus thrombosis but lacking several features of that syndrome are not readily explained, in the writers'

opinion. In the absence of all other causes, however, they were forced to conclude that it resulted from the venous stasis occasioned by the thrombosis centering in the innominate. They mention that this case appears not to accord with the experimental conclusions of Ferrari, working on dogs.

DAVIS, New York.

VISION IN OCCUPATIONAL NYSTAGMUS. L. WEEKERS, Am. J. Ophthal. **3**:162, 1920.

The author draws an analogy between hemeralopia and nystagmus as observed among the soldiers during the recent war, attributing these conditions to muscular and nervous fatigue, and he applies this principle to occupational nystagmus.

He believes forced marches, night work, excessive physical fatigue and the nervous strain of bombardment prepared the field for these conditions, in a similar manner as the poor illumination, unhygienic surroundings of the mine and fatigue do for miners' nystagmus.

He quotes Professor Nuel who insists that two factors destroy the muscular equilibrium usually controlling vision; namely, the failure of the fovea to function and the loss of binocular vision. He explains the first factor by recalling that in dim light the retina sees with the rods and not the cones, and as there are no rods in the fovea it is necessary for the eye to shift its focus 15 to 20 degrees from the fovea for vision. This groping tends to cause the nystagmus of miners, as the lighting is poor and the unhygienic surroundings quickly leads to fatigue. The second factor, the loss of binocular vision, he explains by the fact that the coal has angular and crystalline facets and the lamp lighting these facets is reflected, thus each eye sees different facets, destroying binocular vision, and easily fatiging the eye.

The author believes that miners' nystagmus does not entirely consist of ocular displacements, but includes insufficiency of retinal adaptation, and that dark adaptation is a function not only of the retina, but also of the cerebral centers and as such may be especially affected by over fatigue.

SCARLETT, Philadelphia.

HYPERGLYCEMIA IN MENTAL DISORDERS. F. H. KOOY, Brain **42**:215 (Oct.) 1919.

This paper presents the results of an extended series of observations of the blood sugar of various types of psychotic persons made before and at short intervals after the ingestion of a standard breakfast. The values obtained from twenty normal persons following the same schedule are used as the basis of comparison. In ten cases of dementia praecox, nine cases of dementia paralytica and eight cases of epilepsy no valid differences from normal persons were observed either in the absolute amounts or in the course of the blood sugar curve after eating. In melancholia, however, there appears to be a tendency not only for the initial values to be higher, but also for the blood sugar increase after eating to be greater than in normal persons, nineteen cases being reported. The same phenomenon of higher blood sugar after eating was observed in four cases of neurasthenia or psychasthenia and in six cases of amentia (confusional insanity). The results obtained from five cases of mania were variable. The author attributes the production of the so-called hyperglycemia to the relatively greater emotional reactions occurring in these

types, since the patients showing the greatest degree of anxiety had at the same time the highest amounts of sugar in the blood after eating. He correlates this with an hypersecretion of epinephrin, or better an overexcitement of the sympathetic nervous system, clinical evidence of which is given by the higher blood pressures, the pupillary dilatations, the diminishing reactions, and the reduced intestinal activities obtaining in these patients.

HAMMETT, Philadelphia.

THE "NERVOUSNESS" OF THE JEW. A. MYERSON, Ment. Hyg. **4**:65 (Jan) 1920.

Myerson's masterful presentation of his subject should not pass unnoticed. His reasons for the predilection of the Jew for the psychoneuroses are interesting and convincing. The predisposition springs not from biologic but from "social heredity" which means the influence of groups of life factors handed down for generations in the growth and development of a race. Marked religious differences have given the Jew the stamp of unlikeness and therefore, he has been prejudged as different and unsociable. Further, "he was excluded from all occupations, in the pursuit of which the manual-motor side of his nature might find expression." This conditioned a dislike for the occupations from which he was shut out and gave him an inferior physique. By force of circumstances he developed largely into an urban, sedentary, cerebral type. Living in constant dread of ridicule, degradation and massacre, a lowered threshold for fear stimuli was acquired. The effect of this emotion on the material organism must also be considered. In this connection it is often stated that the Jew is particularly liable to arteriosclerosis and diabetes. In favor of social as against biologic heredity, the author cites the rapid amalgamation of the Jew with his Gentile fellows when conditions are favorable and the character and habit modifications that this amalgamation effects.

STRECKER, Philadelphia.

THE LIMITATIONS OF TINEL'S SIGN IN PERIPHERAL NERVE INJURIES. BYRON STOOKEY, Neurol. Bull. **2**:380 (Oct.) 1919.

Stookey discusses both the importance and the limitations of Tinel's sign, signe du fourmillement, which is obtained by pressing on or tapping the distal stump of a nerve trunk containing regenerating axons, and describes three cases to illustrate various points. The sign in Stookey's opinion has only qualitative value and is very important when taken in conjunction with other signs of regeneration but furnishes little guidance for operation when taken by itself, as its presence does not preclude the existence of complete interruption. The sign, according to Stookey, is found (1) in cases where continuity exists and the neuraxes can penetrate into the distal stump, (2) when despite more or less complete interruption a sufficient number of neuraxes penetrate the distal stump, but the interruption is sufficient to prevent ultimate functional return, and (3) when the interruption is absolute but a few straggling neuraxes, either from the proximal stump or some of its adjacent branches, find their way by a devious path into the distal stump. The sign, therefore, even though it be present and progressive should not be considered an evidence of satisfactory regeneration unless, when the anatomic field is known, confirmatory evidence is also found. In so far as it indicates quali-

tatively a downgrowth of neuraxes, it is a valuable sign; when it is progressive and is associated with a recession of the field of sensory loss, etc., it is indicative of regeneration. Alone it is not capable of quantitative determination and is devoid of definite significance.

WECHSLER, New York.

EPIDEMIC ENCEPHALITIS AND KATATONIC SYMPTOMS. EARL D. BOND, Am. J. Insan. **76**:281 (Jan.) 1920.

The author emphasizes the katatonic aspects of five interesting cases. Three were undoubted examples of epidemic encephalitis; the fourth was an acute hemorrhagic encephalitis, and the fifth a katatonic episode in the course of a chronic depression. Bond's paper is a distinct addition to the literature of katatonia and his observations are added proof in favor of its organic nature. The evidence cited should make one realize the futility of attempting to find a purely psychogenic origin for this symptom. That its occurrence, in some instances at least, may be a matter of fleeting but none the less actual brain involvement, seems to be a reasonable and plausible hypothesis.

Attention is called to our vocabulary deficiency in the description of tonal muscle abnormalities. Much valuable clinical evidence must be lost because we cannot or rather do not suitably reproduce on paper the particular variation of the symptom observed at the time of examination.

STRECKER, Philadelphia.

Society Transactions

CHICAGO NEUROLOGICAL AND THE CHICAGO SURGICAL SOCIETIES

Joint Meeting, Jan. 2, 1920

CHARLES E. KAHLKE, M.D., President of the Chicago Surgical Society, *in the Chair*

SURGICAL ASPECTS OF PERIPHERAL NERVE INJURIES. Presented by DR. DEAN LEWIS.

I prefer to demonstrate this evening some of the results that may be obtained in peripheral nerve surgery, rather than to discuss formally the many problems presented in injuries of this type.

CASE 1. — This patient was wounded Oct. 27, 1918. He sustained four wounds. The internal condyle of the left femur was fractured and subsequently removed. As you see, there is a large scar over the left shoulder posteriorly and also one over the sacrum. The fourth wound is directly over the course of the lower part of the right musculospiral nerve which was evidently divided, for paralysis was noted immediately. On Feb. 21, 1919, the musculospiral nerve was exposed in the groove between the brachialis anticus and the supinator longus. The nerve had been completely divided. The neuroma on the end of the proximal segment was resected until healthy neurofibrillae were exposed and the scar tissue of the end of the distal segment was also removed. Two tension sutures of fine catgut were inserted and the epineurium was then closed with fine chronic catgut.

Definite evidences of return of motion were noted about five and one-half months after suture. As you can see, there is now almost complete return of power in all the muscles supplied by the musculospiral nerve. The extensors of the fingers react slowly. The patient states that he cannot throw a ball as he cannot release the fingers rapidly. Marked hyperesthesia of the skin supplied by the musculospiral nerve was noted during recovery. This has gradually subsided.

CASE 2.—This boy was wounded during the first week of November, 1918. The right musculospiral nerve was divided by a machine-gun bullet which perforated the arm, apparently passing through the humerus without fracturing it. The nerve was sutured on March 17, 1919. He has complete return of motor function in the radial extensors and also some in the common extensors of the fingers. There is no evidence of return of function in the abductor of the thumb. Regeneration has occurred through a neuroma. At the point of suture there is a definite fusiform enlargement of the musculospiral nerve which is painful on pressure. Regeneration has occurred rapidly, however, as evidences of return of motion were noted five and a half months after suture.

CASE 3.—This boy was wounded in the Argonne Forest. The musculospiral nerve was divided on the inner side of the arm just before it entered the groove. It is somewhat difficult to suture the nerve in this position, as it lies

somewhat deeply and bleeding from the superior profunda artery may be troublesome. The operation was performed on April 22, 1919. Motor function is present in the radial extensors. The power is good. There is some evidence now of return of motion in the extensor communis digitorum. The abductor of the thumb is still paralyzed.

CASE 4.—This patient presents some interesting features. He was wounded in the Argonne Forest, sustaining a gunshot fracture of the humerus near the middle and an injury of the musculospiral nerve. The wounds were completely healed and a roentgen-ray examination of the humerus was, therefore, not made. When the incision to expose the divided nerve was made, a sequestrum surrounded by granulation tissue and some pus was found in a cavity directly beneath the divided nerve. The sequestrum was removed, the granulation tissue curetted out and the cavity iodized. An end-to-end suture of the divided nerve was performed and the wound closed. Much to my surprise the wound healed by primary union. Up to ten days ago there has been absolutely no evidence of return of function. Suddenly the patient noted that he could extend his wrist. Since then the improvement has been rapid. Sudden return of motion, as in this case, has been noted in other cases that we have observed.

CASE 5.—This patient had a combined median and ulnar lesion. Both nerves were divided in the middle of the arm. You can see the large scar that followed the healing of a wound caused by a piece of high explosive. At the time he was operated on there was a distinct lymphedema of the skin below this partially constricting scar. At the time of operation conditions seemed unfavorable because of the lymphedema and the difficulty of making an end-to-end anastomosis. The suture was made under some tension. I believe that the results are surprisingly good, for the patient has regained almost complete use of the muscles supplied by the median nerve and there is some return of motion in those supplied by the ulnar. One of the most striking changes is the appearance of the hand; the color is better and there has been a decided improvement in the general appearance.

CASE 6.—This patient was wounded on September 22. He received a high explosive wound of the left foot and one of the posterior aspect of the left thigh. About 3½ inches of the sciatic nerve were destroyed. He was operated on March 19, 1919. An end-to-end suture was performed after the segments of the sciatic nerve were mobilized and the knee flexed to a right angle. About one and a half months ago power returned in the muscles supplied by the internal popliteal nerve. The muscles supplied by the external popliteal are still paralyzed. There has been decided improvement in the appearance of the leg and foot. Both appear larger and are more solid. The atrophy is much less marked.

The next two cases are instances of recovery or improvement after neurolysis. The first patient received a high explosive wound. The left humerus was fractured and the left musculospiral nerve injured. You can see that there is considerable deformity. According to the clinical findings, the musculospiral nerve was completely divided. On April 26, 1919, the musculospiral nerve was exposed. When dissected out of the scar, it had a frayed appearance, but four funiculi were found passing from the proximal to the distal segment. This patient has now extension of the wrist and beginning extension of the fingers. There is, however, no return of power in the abductor longus pollicis.

The second patient was wounded October 5. According to the symptoms, there should have been found a complete division of the sciatic. When the nerve was exposed on July 30, 1919, it was found embedded in scar tissue, but there was no anatomic division. The nerve was dissected out of the scar tissue and a muscle neurolysis performed. The internal popliteal part of the great sciatic presents evident signs of recovery, but there is no improvement in the external popliteal. It might be argued that in these two cases recovery would have occurred if no operation had been performed, but I do not believe that any one can deny that there is a very definite relationship between the operation and recovery.

The cases presented demonstrate that the musculospiral nerve recovers rapidly after suture. The median, ulnar and internal popliteal also recover fairly rapidly. The external popliteal recovers slowly. After sciatic suture, return of function of the muscles supplied by the internal popliteal is noted much earlier than of those supplied by the external popliteal. This is somewhat surprising as the external popliteal is usually easily exposed and the suture easily made. The large amount of muscle that has to be reinnervated might account for the slow return, but the same amount or more has to be reinnervated after suture of the internal popliteal nerve.

End-to-end suture should be performed when possible, for it is the only procedure that gives a high percentage of successes. Mobilization of the segments, displacement of nerves and proper posture will often enable one to make an end-to-end suture, even when at first it seems impossible. The suture material apparently makes no difference. We have employed epineural sutures of fine silk and in other cases have employed transfixion sutures of fine chromic catgut. It is essential to secure as accurate apposition as possible of the nerve ends and to close the epineurium carefully, so that the neurofibrillae will be prevented from straying into the surrounding connective tissue.

TRANSPLANTATION OF PERIPHERAL NERVES. By Dr. G. Carl Huber, Ann Arbor, Mich.

Owing to a clerical error, an abstract of Dr. Huber's remarks appeared in the abstract department, page 437, of the April issue of the Archives.

CLINICAL SIGNS OF PERIPHERAL NERVE INJURY AND REGENERATION. Presented by Dr. Lewis J. Pollock.

Dr. Pollock described such signs of nerve injury and regeneration as appeared to him to be most important from the study of cases coming under his direct observation.

He called attention to the influence of supplementary movements on the clinical picture of paralysis (Arch. Neurol. & Psychiat. **2**:518 [Nov.] 1919). He then described the influence of nervelap on the relatively early return of sensation to pin-prick (Arch. Neurol. & Psychiat. **2**:667 [Nov.] 1919). He was unable from his experience to differentiate anatomic from physiologic interruption, and stated that in general the course of the clinical picture is much more important than any group of signs for the purpose of determining the severity of a lesion. Among the signs of a severe lesion he found that analgesia to severe pinching occurred in that area insensitive to pin-prick occupying the isolated sensory supply of the nerve. He stated that absence of demonstrable atrophy some months after injury is not an indication of a reparable injury. Absence of pain when the trunk of the nerve is subjected

to pressure below the seat of the lesion was demonstrable in many severe lesions. On the other hand, not a few irrecoverable lesions showed the preservation of pain to such pressure. Absence of pain to pressure on the paralyzed muscles was likewise found to be an unreliable sign. Trophic changes could be employed as an indication of the severity of the lesion only when judged in the light of the presence of other signs. This was particularly true of ulceration which occurred only in analgesic areas, cessation of growth of nails in immobilized extremities and atrophy of bone under similar conditions.

Many cases of complete so-called physiologic interruption of a nerve showed their first sign of regeneration at such a time as one would expect it to occur, had the nerve been divided at the time of injury and sutured. It is reasonable to assume that in this type of severe lesion complete descending degeneration had occurred and conditions permitted the subsequent regeneration of the axon. Return of pain on pinching of the skin was frequently the first sign of nerve regeneration. Spontaneous aching, and more frequently a sensation of a "different feeling," were found. Tinel's sign had not been found to be of any precise value.

Relative to the return of sensation, only when that portion of the skin representing the isolated supply of a nerve becomes sensitive can it be said that regeneration is present. Under this condition at no time did protopathic sensibility return before epicritic. Arrest of atrophy and return of tonicity in those patients recovering some months after injury are not valuable as signs of regeneration. Following resection and suture, reaction to faradism did not return before motion. In patients recovering a number of months after the injury, electrical reactions were not of great value in the determination of regeneration. In the recovery consequent to resection and suture, the various peripheral nerves showed a distinctive picture in the relatively constant progression of recovery of the several muscles supplied by them.

DISCUSSION OF REPAIR OF PERIPHERAL NERVE INJURIES

DR. ERNEST SACHS, St. Louis: I do not feel that I can add to this subject. In addition to hearing the papers this evening, I also had the good fortune this afternoon to see Dr. Huber's beautiful specimens. They added tremendously to the interest of what I have heard tonight, and that has cleared the atmosphere remarkably on the subject of nerve surgery. After a study of this large amount of material, both clinical and experimental, certain things long thought and taught about peripheral nerve surgery, can be discarded for all time. The various old methods of trying to bridge defects by making flaps, and so on, have now been shown to be unsatisfactory. With proper methods peripheral surgery and peripheral regeneration are perfectly feasible.

Many of you may remember—I think it was three years ago—that at the meeting of the Clinical Congress in Chicago we heard some pessimistic remarks from Sir Berkeley Moynihan on the subject of peripheral nerve surgery. Undoubtedly these pessimistic reports were due to the fact that the fundamental principles of nerve suture as now known were not followed at that time: first of all, exposing the fibrillae in the proximal and peripheral stumps; second, a dry field; third, doing whenever possible, primary suture or direct suture and closing the neurilemma sheath so that connective tissue will not grow in.

One other point that was not emphasized, but which should be covered, is that the handling of nerves is very different from the handling of other tis-

sues of the body. A nerve must be handled with great gentleness. Another thing which my experience has shown to be very important is to keep the nerve warm, and not allow it to become chilled.

The only phase of the work on which I have had any practical experience confirms absolutely what Dr. Huber said. It was first described by Rehn who emphasized its great value in applying it over defects in the brain. About eight years ago I tried that out experimentally and became convinced that his observations were absolutely wrong; that the fat was completely replaced in a short time by a dense mass of connective tissue, and several cases to which I applied this clinically, I had an opportunity later on of again opening and found the same thing occurring. The fat is replaced by a dense mass of connective tissue.

One point that Dr. Pollock brought out; namely, that it is impossible to tell whether a nerve is anatomically or physiologically divided, seems to me a valuable contribution. What he said about the length of time after which a nerve may still regenerate, I do not question, but his statement might lead to faulty surgical treatment. If it is possible for a nerve to regenerate seven or eight months after an injury, I do not believe it is advisable to wait as long as that to determine whether or not the nerve is absolutely divided. If Dr. Pollock believes that, I must take issue with him. If there is evidence that the nerve is completely blocked, I believe the proper procedure is to explore the region, determine the nature of the block and, if necessary, take steps to correct it.

I do not wish to discuss his statements concerning the time of recovery of a nerve because he has had much material to work on, but anyone who has busied himself for a long time with neurology cannot help feel a little disconsolate, as we all do, when something we have banked on for many years has been undermined; in other words, certain props have suddenly been knocked from under one. I refer particularly to what he said about the work of Head in regard to peripheral nerve disturbance. His evidence is extremely interesting; it is difficult to contradict; but he has not absolutely refuted the work of Head and his various co-workers which extends over many years. I do not quite see how he explains the return of sensation in the ulnar anesthetic area by the overlap, for it seems to me that if the overlap controls a large part of that area, the sensation of that area—that overlap—ought to be present immediately after the injury. In other words, the only anesthetic area should be the exclusive area supplied by the ulnar nerve. As I understand his observations, and I have read his article on the subject, there is a complete loss of sensation, and after a short time some sensation returns in that area. I cannot quite see why there should not be only the exclusive area supplied by the ulnar nerve as he has worked it out, and from the beginning. I hope Dr. Pollock will answer that point in his closing remarks.

Dr. G. Carl Huber (closing on his part): I have enjoyed listening to the presentation of the neurologic side of this subject by Dr. Pollock, and was personally very glad to hear the questions asked with reference to interpretation of facts presented, because there has not been unanimity of view on questions under discussion. Referring to Dr. Pollock's work, in the first place, there are no hard and fast boundaries of peripheral nerve distribution. I think the anatomists will have to change their figures—at least for so-called exclusive areas of peripheral sensory nerve distribution. I think Dr. Pollock has helped us a great deal in that direction, and I can readily see that there

may be an apparent overlap in sensory areas, more than is generally recognized. The sensory nerve supply for a particular area, for instance, the peripheral ulnar field, need not be exclusively supplied by the ulnar; other bordering nerves may be educated in the course of a few weeks to give perception from the borders of the same area. I am confident that it is not necessary in practical work to lay undue stress on Stoffle's studies on the specific funicular structure of nerves. Any one who has worked experimentally with regeneration of nerves realizes that from ten to fifty new neuraxes may bud toward the periphery from a single central neuraxis, and fifteen or twenty new neuraxes are often found centrally in a single old neurilemma sheath. No matter how carefully primary suture is made, there is a great tangle of these new nerve fibers as they pass through the connective tissue of the wound, and especially is that the case with secondary sutures. A large number of fibers pass from the stump along the transplant in the connective tissue surrounding the transplant. I am sure sensory nerve fiber branches reach the motor nerves, and that central motor nerves reach the distal sensory nerves and are maintained for a time. They make no distal connection and in time degenerate. I am confident there is never complete regeneration of the peripheral stump, and to some extent there needs to be reeducation of the nerve centers after every regeneration of the peripheral stump. The anatomic findings often give very distinct motor and sensory recovery without full functional return.

Dr. Pollock (closing): I did not wish to be understood as saying that no operative procedure should be undertaken in the case of a severely injured peripheral nerve in less than eight or nine months. The point I wished to emphasize was, that if such an injured nerve can at times recover spontaneously, if any surgical interference be attempted it should not be in the nature of resection and suture.

I cannot thoroughly discuss the question of nerve overlap at this time. However, I might point out several facts. First, while the work of Head and his co-workers has been accepted largely by physiologists and neurologists, not all recent publications of physiologists confirm their findings. We need only refer to the work of Boring which proves definitely the educability of overlap nerves. If three nerves supply the entire leg below the knee and we cut two of them, the third is left. If, therefore, we have a lesion of the internal saphenous and the external popliteal the sensibility present is subserved by the internal popliteal. If we have a combined lesion of the internal saphenous and internal popliteal the external popliteal is left. By using this method of residual sensibility we can reconstruct the distribution of the total sensory supply of the various peripheral nerves. I maintain that the early return of sensibility to prick pain occurs only in such areas as are within the areas of nerve overlap of adjacent nerves. These areas constitute that part of the residual sensibility of a peripheral nerve which extends beyond the limit of the anatomic sensory distribution of an adjacent nerve. If this early return of pain sense always appears in areas of nerve overlap; if it never appears in such an area when the nerve supplying this area with sensation is cut; if, when it has appeared and the adjacent nerve is subsequently severed, it disappears, and, finally, if it has appeared and resection and suture of the originally injured nerve have no effect on this sensibility, it seems to me that my point that this early return of sensibility to prick pain is due not to nerve regeneration but to nerve overlap, has been proved.

CHICAGO NEUROLOGICAL SOCIETY

Regular Monthly Meeting, Feb. 19, 1920

MYOTONIA ATROPHICA. Case presentation by Dr. Ralph C. Hamill.

The patient was a man who gave a history of difficulty in relaxing his grip. In military service he did not handle his gun well. One brother and one sister were normal. The muscles were good with the exception of the shoulder girdles and forearms. The trapezius was rather prominent above the scapular outlines, which were abnormally prominent because of the small size of the supraspinatus. The extreme atrophy of the sternocleido mastoids also emphasized the prominence of the trapezius. The upper deltoids were somewhat wasted; there was increased prominence of the lower portion of the vertebral border of the left scapula; the pectorals were quite good. The flexor muscles of the forearms were wasted, especially the left, the patient being left-handed. Flexion and extension of the wrists were weak, especially the left. The grip was poor, mostly exerted between the thumb and last phalanges of the fingers. Flexion of the hand was poor on the left, fair on the right. The thumb muscles were involved. There was no marked atrophy in the hands. Pronation and supination were weak; abduction and adduction of the fingers was fairly good; he could not make a fist. Flexion of all phalanges was nil or nearly so. Apposition of the thumb was weak. There were no atrophies in the lower extremities.

Reflexes: Plantar reflexes were normal; the right ankle jerk was absent; the left was present only with reinforcement. The right knee jerk was diminished, the left less so. Abdominal reflexes were normal, wrist jerks were absent, the left triceps was present, the right was absent except with Jendrassik. The jaw jerk was absent. The eyelids did not close completely. Movements of the forehead were very slow; those of the lips of small amplitude; he could open his mouth well and the tongue protruded without tremor. After biting hard he had difficulty in opening his mouth, but after once opening it there was no further difficulty. Strength of the masseters was normal, as was also movement of the eyes. Sense of passive motion was slightly diminished in the toes; normal in the fingers. The sternocleidomastoid muscles were almost absent. He could hop well on either foot. There was complete absence of fibrillary or other twitching. After gripping firmly he had difficulty in relaxing or in extending the fingers, but after exercising a few times he could relax them more easily. As bearing on the possible glandular origin of his trouble, it may be noted that the testes were undescended.

DISCUSSION

Dr. G. B. Hassin asked whether any myotonic reactions were present.

Dr. Hamill replied that he could not determine any myotonic electric reaction. The case was interesting because the disease was unrecognized in the Army and the man was accounted a serviceable soldier.

FURTHER OBSERVATIONS ON THE PRESENCE OF ARSENIC IN THE SPINAL FLUID. Dr. George W. Hall.

This paper is published in full in this issue.

DISCUSSION

Dr. H. I. Davis asked whether the experiments had influenced Dr. Hall's decision as to the use of neoarsphenamin.

Dr. Julius Grinker asked whether Dr. Hall had made comparisons in the amount of arsenic found in the fluid following the intravenous as compared with the amount found following the intraspinal injections.

Dr. Ralph C. Hamill asked whether Dr. Hall had considered leukocytosis as evidence of inflammation of the membranes, and whether the inflammation was not itself curative.

Dr. A. B. Yudelson asked whether Dr. Hall had noted the particular type of cerebrospinal syphilis in which these fibrins were observed.

Dr. Hall, (closing) in reply to Dr. Grinker, said that he had tested the spinal fluid for arsenic several times after intraspinal injections and had concluded that it was a dangerous and inefficacious procedure. He did not feel able to answer Dr. Hamill's question.

As to whether the results were just as good from injecting the autoserum as those of the Swift-Ellis method, he had no definite data, but he believed that the clinical results were equally good. The reactions were very different in different persons when the autoserum was used. Such marked differences of reaction were not observed, in the use of intraspinous mercury. If the results depended entirely on irritation of the meninges he was inclined to think that the mercuric chlorid or mercuric succinimid produced a more uniform reaction, although more painful. One one-hundredth grain of mercuric succinimid or mercuric chlorid could be safely used, but $\frac{1}{50}$ or $\frac{1}{25}$ grain was hazardous.

BRAIN ABSCESS WITH PRESENTATION OF GROSS SPECIMENS.

Dr. Peter Bassoe.

In Dr. Bassoe's absence, Dr. Hassin presented specimens and related the histories of the following cases:

1. *Acute Traumatic Brain Abscess.*—A child, 17 months old, received a superficial wound 1 cm. to the right of the median line at the vertex. A week later pus appeared and two weeks after the accident the child was admitted to the hospital with no symptoms except discharge from the wound. Roentgen-ray examination revealed a small circular perforation of the skull at the site of injury. Fever gradually developed and three weeks after the injury the wound was explored. The dura protruded through the perforation of the skull. On aspiration pus was obtained and drainage established. The child died two months later and the abscess was found to extend almost to the occipital pole posteriorly and a short distance into the frontal lobe anteriorly.

2. *Chronic Frontal Lobe Abscess Following Gunshot Wound.*—A man, 21 years old, was admitted to the hospital in a semicomatose condition, moaning with pain in the head. He had bilateral choked disks, slight fever, leukocytosis of 14,900 and a discharging sinus in the right frontal region. It was learned that he had been in good health for years until the morning of the day of admission, also that eight years previously he had sustained a gunshot wound of the right side of the forehead. He died twelve hours after admission and the necropsy revealed a large thick-walled abscess which occupied the anterior half of the right hemisphere. The dura was adherent both anteriorly

and externally, and beneath the latter point of adhesion a flattened piece of bullet was detected. Apparently the patient had for eight years harbored an abscess and the bullet fragment without discomfort.

3. *Fracture of Left Temporal Bone with Abscess at the Site of Contrecoup in the Right Frontal Lobe.*—A man, 29 years old, who had been struck by an automobile and sustained a fracture of the left temporal bone with hemorrhage from the nose and ears, developed mental confusion, slight aphasia and weakness of the whole right side, including the lower part of the face. Naturally, a left-sided brain lesion was expected, but on operation six weeks after injury, nothing was found except a small accumulation of clear fluid. The patient improved, but three months later developed purulent meningitis and died. The brain, when obtained by Dr. Bassoe, was in bad condition, but it was possible to determine that in addition to recent meningitis and superficial necrosis at the site of operation, there was a small abscess near the under surface of the right frontal lobe surrounded by necrotic brain substance and the overlying pia showed rusty discoloration. There was also a rust colored softened area on the under surface of the left frontal lobe, but this had not suppurated. It was thought that the hemorrhage and necrosis at the site of contrecoup in the frontal lobe were the direct result of the injury and that infection from the blood stream had taken place later. No focal symptoms were attributable to this lesion which involved a silent area. On the other hand, the rather slight lesion of the left hemisphere involving a highly important functional area, caused distinct focal symptoms.

4. *Brain Abscess Secondary to Lung Abscess Following Tonsillectomy.*—A woman, aged 36, had a lung abscess drained three weeks after tonsillectomy under ether. Eleven months later she developed severe headache, vomiting and optic neuritis. The spinal fluid gave a negative Wassermann test, a cell count of 957, a positive globulin and Lange gold test. Necropsy revealed an abscess cavity which occupied the right frontal and temporal lobes.

5. *Cerebellar Abscess with Abscess of Sphenoid Sinus.*—There were the postmortem findings in a girl 13 years old, in whom a diagnosis of brain tumor of undetermined location had been made. There had been slight fever and a leukocyte count of 10,400 prior to a subtemporal decompression, following which there was relief for two days and then increased papillitis, fever and leukocytosis of 16,400. No evidence of sinus disease had been found, either by roentgen-ray examination or other methods. The patient died about six weeks after the onset and two weeks after the operation. There was a walnut-sized abscess in the left lobe of the cerebellum, and the right sphenoid and posterior ethmoid sinuses contained a large quantity of mucopus, which was not under any tension.

(These cases will be found reported in full in the May number of the *Medical Clinics of North America.*)

DISCUSSION

Dr. Julius Grinker said the mortality from brain abscess was high because it was difficult to localize and difficult to drain; because there is a tendency for abscesses to multiply, or for hour-glass abscesses to form. Drainage should be maintained until the area was completely drained. He thought it was the multiple abscesses that killed. In one instance a frontal lobe abscess was diagnosed, operated on and the patient made a clinical recovery; but something was still wrong. The choked disk, previously well marked, had cleared up

for only a short time; indeed, it became more intense than before operation. Undoubtedly this meant that pressure was still present. When the symptoms became more aggravated the patient was again operated on but the surgeons missed the other abscess, found postmortem.

CONTRIBUTIONS TO THE STUDY OF CRUVEILHIER'S "TUMEURS PERLEES." DR. PERCIVAL BAILEY.

Since Cruveilhier, in 1829, described peculiar pearly tumors at the base of the brain, about sixty-two cases have been reported. They occur as about 0.2 per cent. of brain tumors. "Cholesteatoma" is the term usually applied to these growths, which is unfortunate, because often they do not contain cholesterin and moreover congenital cysts, collections in the middle ear after chronic otitis media, etc., contain cholesterin crystals but have nothing in common with these pearly growths.

The favorite location is on the base of the brain, beneath the dura but outside the brain tissue proper. They are occasionally found in the ventricles. Seven cases are described in the fourth ventricle. Grossly, they startlingly resemble mother-of-pearl. The outer portion is distinctly laminated. The interior looks like dry cottage cheese and easily crumbles. The surface peels away from the surroundings.

The microscope reveals four distinct layers which may be termed from without inward, stratum durum, stratum granulosum, stratum fibrosum and stratum cellulosum. The stratum durum consists of an almost homogeneous substance with occasional pyknotic nuclei. The stratum granulosum is made up of flattened cells containing definite nuclei, and granules which stain well with cresyllightviolet, evidently of keratohyalin. The stratum fibrosum consists of layer after layer of homogeneous material containing no evidence of cellular structure. The stratum cellulosum is composed of polygonal cells with heavy cell walls showing only occasionally a poorly preserved nucleus. The interior of the mass is a mixture of broken-down cells and fatty material. Cholesterin crystals are said to be occasionally found.

There are many theories of their origin but the structure is so definitely epithelial that their origin from ectodermal inclusion is generally accepted. They have hitherto been of more interest to the pathologist than to the surgeon. So far as I know, the cases here reported are the first to be successfully operated on.

CASE 1.—Peter Bent Brigham Hospital; surgical number 10302; housewife, aged 28.

Complaint: Headache, vomiting, failing vision, tinnitus and unsteadiness in walking.

Chronology of Symptoms: For five years headache and vomiting. For one year pain in the upper teeth on the right side. For eleven months failing vision and tinnitus. For six months staggering gait. For several weeks weakness and tremor of the left hand and emotional instability.

Positive Neurological Findings: Nasal margin of left optic disk definitely obscured by edema. Slight and poorly sustained nystagmus on looking to right and left. Auditory acuity diminished in left ear. Slight ataxia and adiadokocinesis of left hand on long exertion. Epigastric and abdominal reflexes absent. Gait staggering with perhaps deviation to left. Slight ataxia of left leg. Emotional instability. Caloric tests elicited, slight reactions from the right ear and marked reactions from the left.

Diagnosis: Intracranial tumor involving the cerebellum.

Operation: Suboccipital exploration by Dr. Harvey Cushing. Beautiful pearly tumor exposed completely filling the fourth ventricle, flattening the cerebellum and extending down into the spinal canal. The tumor was completely removed, leaving a smooth, glistening, dilated fourth ventricle.

Postoperative Progress: Patient developed a sixth nerve palsy which rapidly cleared up, and she was discharged a month after operation. When last seen eight months later, she had gained 20 pounds in weight and there were absolutely no neurologic findings.

Case 2.—Peter Bent Brigham Hospital; surgical number 11152; schoolboy, aged 13.

Complaint: Failing vision.

Chronology of Symptoms: For six years severe frontal headaches which lately had disappeared. For eleven months gradual failure of vision in right eye which progressed to total blindness in six months. For six months gradual failure of vision in the left eye.

Treatment Before Admission: Right subtemporal decompression about a year before admission.

Positive Neurologic Findings: Well nourished but not fat, 6.64 kilogram below standard weight for his age and 7.7 cm. below standard height. Hands small but not typically pituitary. Genitalia showed evidence of early adolescence. No signs of dystrophy. Blindness in right eye and temporal hemianopsia in left. V. A. left eye 10/70. Bilateral primary optic atrophy. Slight nystagmus on looking to extreme left. Roentgen ray showed absorption in the frontal region. No signs of hydrocephalus. Basal metabolism, 14.

Diagnosis: Supracellular tumor.

Operation: Transfrontal exploration by Dr. Harvey Cushing. Characteristic pearly tumor about the size of a golf ball in the third ventricle. Removal entirely except for a few small fragments of wall which clung to blood vessels and nerves.

Postoperative Progress: Patient became restless, rolling and tossing constantly, crying and whining incessantly; answered questions only after persistent effort but with perfect comprehension; kept head covered and complained of being cold although temperature remained above normal for nineteen days. The constant involuntary movements of the limbs made it impossible for him to walk. Insomnia was a prominent symptom. This condition disappeared in three weeks. He was discharged well with visual acuity slightly less than before operation.

Histologic examination proved both these cases to be true pearly tumors. (This article will appear in full in *Surgery, Gynecology and Obstetrics.*)

DISCUSSION

Dr. G. B. Hassin said that his examination of Dr. Bailey's slides showed these tumors to be avascular. They probably originated from the mesothelial cells of the pia arachnoid. These cells were also found in the dural interspaces where, according to some authors, they may give rise to tumor formations like endotheliomas. The tumors are benign. It is a peculiar fact that they were never found in the spinal cord or brain tissues proper.

The youth of one of Bailey's patients, 13 years, was noteworthy. The majority of authors assert that these tumors do not occur under 40 years.

He asked whether Chiari's case reported as cholesteatoma of the spinal cord was a real "tumeur perlée" or something else, and whether he had found in the literature a patient as young as his.

Dr. Bailey replied that these tumors usually occurred at the end of the third or beginning of the fourth decade and he thought no case had been reported in a patient as young as 13.

Chiari's report was unaccompanied by histologic examination, hence it was impossible to say whether the growth was a true pearly tumor or not.

THE HISTOPATHOLOGY OF BRAIN ABSCESS, WITH LANTERN SLIDE DEMONSTRATION. Dr. G. B. Hassin.

Published in full in this issue.

DISCUSSION

Dr. Julius Grinker took issue with the speaker on the conclusion he drew from the histopathology of brain abscess as to the value of the Swift-Ellis treatment for neurosyphilis. If Dr. Hall's statement that arsenic had been found in the spinal fluid in 25 per cent. of the cases following intravenous injection were true, the value of the intravenous method was thereby proved—a method which some had considered valueless. However, these findings should not prevent the use of the Swift-Ellis treatment in cases that do not respond to intravenous injections. Some of his results, as well as those of others, had been exceedingly good.

Dr. I. Leon Meyers thought that Dr. Hassin's work showed his statements regarding fatty substances to be true, but this was not absolutely true as regarded every other substance. In Flexner's experiments on poliomyelitis, it had been proved that by injecting the virus into the subarachnoid space of the ape, one could produce the infection, while the cerebrospinal fluid from a patient with this disease proved to be noninfective. It was evident that the virus passes into the brain from the subarachnoid space, but not in the reverse direction.

Dr. G. B. Hassin said he did not intend to discuss the Swift-Ellis method as he believed it had been abandoned by the majority of practitioners. Dr. Hassin's further remarks are covered in the paper.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

The Fortieth Anniversary Meeting, Jan. 15, 1920

George A. Waterman, M.D., *President, in the Chair*

FORTY YEARS' PROGRESS

Dr. Walter Channing

Jan. 6, 1880, when I called the first meeting of the Society to order I little thought that forty years later I would again address it as the only surviving member of the nine then present. That I alone of the nine men should be living supposedly might make me realize that forty years is a great stretch of time.

Fortunately, if the men themselves have gone, we still have the inspiration and comfort of what they stood for and accomplished and younger men to carry the work forward. In 1880 state hospitals in most cases provided good accommodations for their inmates, but in the full sense of the word were not hospitals. They were managed by men who were members of the "Association of Medical Superintendents of American Institutions for the Insane." When an effort was made to change the name in the late eighties to the more modern one it bears at present, the offer was refused in indignation. Not scientific development, but the housing and providing for inmates good care at the lowest per capita cost was the grand object to be achieved. Proper medical attention was regarded as necessary, but careful clinical study of the patient was rare. Laboratories existed for routine pathologic work, There were no training schools for nurses. Mechanical restraint was general and regulated only by the individual superintendent.

In 1879 Dr. Folsom was lecturing on mental diseases in the Harvard Medical School, and in 1883 published a small book for students entitled "Mental Diseases." This book gives a good idea of what might be considered rather progressive ideas for the time. Incidentally it is stated that "in the United States census when there is doubt in the matter the individual is classed as an idiot if the disease occurred before the age of 12 in girls and 14 in boys, and as insane (demented) if above that age."

In 1879 Dr. Jelly left the McLean Asylum and was succeeded by Dr. Cowles. Up to that time the McLean Asylum labored to be a comfortable house for the mentally diseased. The patients were called "boarders" in an effort to make them forget they were under restraint, but the advent of Dr. Cowles meant a step forward. A process of scientific and medical housecleaning began at the McLean which never ceased until he laid down his badge of office, and it was a sad day for Massachusetts and progress in mental diseases when he did so.

I am simply calling attention to a few of the factors which have constituted some of the links in the chain of progress and some of the strongest of these links were forged by Dr. Cowles. He designed the present McLean buildings which have been an object lesson for the whole country. He established laboratories not simply for the study of pathology, which up to his time had been held in leash, but by the deepest and broadest methods the effort was made to solve problems which hitherto had been unfathomable.

The hospital which had now been changed from an asylum underwent changes in administration. The first training school for mental nurses was established and the patients treated as mental cases.

Dr. Folsom was appointed on the Commission by Governor Wolcott in 1896 to consider the then existing methods of supervising the insane. This commission on which Dr. Folsom worked faithfully and energetically may be regarded as the starting point for the care of our insane. This society worked indefatigably, and in 1900 a state board of insanity was appointed.

Our Society was organized at a most fortunate time for the period of stagnation in the study of mental diseases was nearly at an end. Curiously enough, the Guiteau trial in 1882 was a factor in turning the tide. The experts for the prosecution presented a dead wall of conservatism with hardly a chink in it to admit a ray of the light of modern ideas. On the other hand, some of the experts for the defense were reviled and laughed at for some advanced ideas. After Guiteau's execution these ideas rose up and walked abroad un-

comfortably like ghosts. It was proved that they were correct and clearly seen that Guiteau was insane, and the subject of a definite form of mental disease. Of course, the Guiteau trial was merely a sign of a general awakening.

Dr. Cowles and other men with advanced ideas needed a forum wherein to present them for discussion and correction. Our Society was intended partly for this purpose. Another most important object was to back up any reform in the care of the mentally ill and we had wonderful opportunities which were largely furnished by the State Board of Insanity through Dr. Copp. He was another of our leaders imbued with what we might call genius. He was constructive in ideas and execution and carried to a successful conclusion reforms and improvements considered impossible.

The greatest thing Dr. Copp did while Secretary of the Board was to work out a plan for the better care and management of our insane. The state care act had been passed and with Dr. Copp acting in cooperation with the Board of Insanity, the backing of this Society and the endorsement of the Massachusetts Medical Society it was possible to impress on the legislature the necessity of going further in the development of our institutions and care of the insane. The Boston Lunatic Hospital was taken over by the State and finally, as the biggest thing of all, the Psychopathic Hospital was built.

What that hospital has accomplished under the influence of the master mind of its director is known, in some measure at least, to us all. The results have been epochal. They have changed an almost unknown branch of medicine into a dignified, scientific medical specialty. They have systematized a mass of disorganized knowledge and in so doing have added greatly to that knowledge. They have improved on treatment and have demonstrated that mental diseases can be cared for as other diseases are in general hospitals, but the greatest result of all has been in the successful efforts of the psychopathic hospital to reach back to the community to attack causes and prevent them. This has been accomplished through the out-patient department. It is evident from the statistics that a large number of persons have been kept out of the state hospitals and many cured before disease has developed. Furthermore, a great fund of knowledge about mental and nervous conditions has been disseminated among patients and their friends.

In time it will come to be seen that an outpatient department like that at the Psychopathic Hospital is of even greater medical and economic value than the hospital itself and an enlightened state will insist on establishing such departments as a means of saving many by diminishing mental diseases.

We cannot use the word "progress" in reference to the period of our Society's existence without thinking of feeblemindedness. Forty years ago it was a neglected and disagreeable subject. Dr. John P. Gray at a meeting of the Association of Medical Superintendents of American Institutions for the Insane in 1884 said: "When we turn to a state like Massachusetts in her philanthropic purposes and see the Governor (Butler) in his annual message declare that the idiots should not be educated because they then come to the realization of their duties and responsibilities which they cannot discharge and that such realization must make them unhappy and that chronic insanity needs no medical care, we might indeed ask if the world was progressing or retrograding."

Think of the change. It is our good fortune to number among our members the man who has been most instrumental in bringing about this great change. The world will rise up and call Dr. Fernald blessed.

Mental hygiene, an entirely new branch of humanitarian work, was unheard of in its present sense forty years ago; it is a wonderful correlation of both social and medical forces, taking advantage of anything and everything, being luckily unhampered by traditions. The National Mental Hygiene Committee in the war will long be remembered. While our Society has not played a leading part perhaps in the Massachusetts Mental Hygiene Society it has contributel some members who have done work which has been really responsible for its success. I would refer in particular to Dr. Stedman.

In time psychiatry will become the most important branch of medical science, including as it does in its scope the moral and physical welfare of the whole human race.

Great progress I believe has been made in our conception of the classification of mental disease. The "key" plan of Dr. Southard can be of great use to the student as he says the plan gives hints for a method of aiming at an eventual diagnosis. It does not prescribe the names of diseases. I believe that we are learning, partly as a result of the war, that labels cannot be pinned on to things as fluctuating as mental conditions. The word "entity," used in regard to mental diseases is a bad one as it implies the idea of fixation. Therapy should always by suggestion indicate that motion must be continuous. Classifications are not of much use if they do not help to keep the patient's mind going along lines which may lead to health, and they are mistaken and therefore pernicious if they convey an idea that a man, by reason of disease, has "lost his mind" and for the time being at least has lost his personality, and become a different kind of being. In the deepest and truest sense mental disease is a result of disturbed social relations of some kind. This is the thing to emphasize when we study treatment and prevention, and it is clearly my opinion outpatient and mental hygiene work which carries us back of the realm of disease and medicine means the next great step in psychiatry.

It is with the greatest satisfaction that one sees on all sides the tendency to drop the word "insanity." We know that it is not a disease but a condition the result of disease, which may affect a person's social status.

I am proud of what has been accomplished in Massachusetts during these recent years, but have fears for the future. In public work requiring the expenditure of money, narrow-minded, short-sighted politicians or managers of institutions often are more influenced by the commercial than the humanitarian aspects of the situation. They must be fought to the last trench unless our progression is to be turned into regression. Here our Society can get in its work. If the hospital policy of the state becomes reactionary, psychiatry is sure to suffer. Eternal vigilance only can prevent it. At our twenty-fifth anniversary I said: "If a medical society has something to do as well as to say, the chances are that it will not only be more useful but will stand a better chance of an active existence." This is as true today as in 1905.

IN MEMORY OF FORMER MEMBERS

Dr. Edward W. Taylor

Dr. Taylor spoke briefly of the activities of the members who had died since the thirty-fifth anniversary, which was held in the same club dining room in 1915. He spoke particularly of Dr. Henry C. Baldwin, Dr. James J. Putnam and Dr. August Hoch.

ENDOCRINOLOGIC ASPECTS OF SOME NEUROLOGIC CONDITIONS. Presented by Dr. Walter Timme.

THE MODERN SPECIALIST IN UNREST

Dr. Southard spoke of the social conditions with which we are confronted in these postbellum days. He emphasized the fact that many of the labor troubles are attributable to leaders of a certain mental type rather than to insurmountable social complications. He spoke of the development in social fields of psychologic and mental examinations and what tremendous aid can be gained in industrial problems when the right men are fitted into the right situations. He felt that the development of psychiatry and psychiatric social service would open unlimited fields of service in the community and the nation. Just as specialists in accountancy and economic efficiency have developed, he felt that a field for the psychiatrist in promoting efficiency along lines involving psychiatric problems was developing.

PHILADELPHIA NEUROLOGICAL SOCIETY

Jan. 23, 1920

J. Hendrie Lloyd, M.D., *President, in the Chair*

STUDIES ON A CASE OF HYPOPITUITARISM. Presented by Drs. E. A. Strecker and H. S. Newcomer.

Family History.—The father of the patient was intemperate; the mother alcoholic, immoral and possibly feebleminded. One sister, aged 4, was "backward," while three brothers and one sister appeared to be normal. Neither of the parents nor any of the siblings showed unusual physical characteristics and were of average stature and weight. An almost indescribable condition of squalor, filth and utter neglect was found in the home. The patient was then 10 years old and had never attended school.

Personal History.—When the patient was 10 years and 3 months old, the height was 61 inches, the weight 109 pounds; ten months later she weighed 165 pounds, had gained 2 inches in stature; the waist measured 33 inches and the bust 38 inches.

June, 1918: "The girl's appetite is enormous. She consumes half a loaf of bread at one meal and requires several helpings of everything on the table. She is gluttonous in her manner of eating, forgetful, and sluggish in her movements."

April, 1919: "Growing very rapidly, appetite is gluttonous."

October, 1919: "Appetite abnormal and bolts whatever is given her; very sluggish in all her movements; likes to go to bed early and sleeps late and soundly."

During the two months prior to her admission to the hospital there was evidently some improvement. The patient became somewhat brighter and more interested, but was still slow, plodding and indifferent.

Physical Examination.—At the time of the physical examination the patient's actual age was 12 years, 2 months; apparent age about 17 years. The height was 63 inches; weight, 155 pounds; head rounded, circumference 22 inches. The profile showed slight maxillary prominence. The nose was somewhat saddle-shaped and short. The ears were large with fleshy lobules. The teeth showed moderate spacing with protrusion of the upper central incisors. The neck was short—girth 14 inches. The hands were wide and relatively short with a tendency to slight finger tapering. The shoulders were rounded

and fairly broad. The chest was broad and deep—circumference 37 inches above and 38.5 inches over the breasts, which were well developed, firm and globular with clearly defined pigmented areola about the nipples. The pelvis was straight with a tendency to the masculine type—circumference at hips, 40 inches. Upper extremities were feminine in type—girth of the arm was 11 inches, forearm 10 inches. Lower extremities were of the feminine type—girth of the thigh was 25 inches, calf 14.5 inches.

The anthropologic measurements on the skeleton were: total heights, 156 cm., height of the acromium process above the floor 130 cm., the proximal end of the radius 97.5 cm., the styloid of the radius 77 cm., the tip of the finger 59.5 cm., the umbilicus 97.5 cm., the anterior superior spines 94 cm., the upper border of the symphysis 85.5 cm., the proximal end of the tibia 46.3 cm., the internal maleoli 6 cm. The interacromial distance 33 cm., interanterior superior spinous 21 cm., and the span 164 cm.

Hair: The head hair was medium fine, dark brown, rather scant and growing low over the forehead and in the temporal region. The eyebrows were scant. There was no other hair growth except a small amount on the labia majora and a scarcely perceptible down on the lower limbs. December, 1919, the menses appeared for the first time.

The skin was dry, warm and slightly roughened and thickened.

Adipose Tissue: There was a large amount of firm fat, more or less evenly distributed with some excess in axillae, breasts, over abdomen and in the gluteal region. The fat had no myxedematous characteristics.

The temperature was continuously subnormal, often as low as 96 degrees. The pulse, respiration and all other examinations yielded normal results. The roentgen-ray report showed an approximately normal sella. The radial epiphyses were open.

Eye examination showed normal fundi; some constriction of the form field especially for the left eye and the slight overlapping of the color fields.

There were no psychotic symptoms. The mental age was 7.5 years, being a retardation of 4.5 years.

The patient while not active or alert was neither dull nor sluggish. She showed a fair degree of interest in various occupations and did well under supervision. She now ate only an average amount. As much as 400 gm. of sucrose was given without producing glycosuria and with a 40 mg. fall in the blood sugar; 325 gm. of glucose did not result in a glycosuria and produced a fall of 15 mg. in the blood sugar. The following experiments were made to show the relationship of the sugar tolerance to the secretions of the endocrine organs. The ingestion of 225 gm. of glucose with the simultaneous subcutaneous injection of 2 c.c. of pituitary extract produced within an hour a blood sugar rise from 128 mg. to 256 mg. per 100 c.c., and an output of sugar in the urine of 0.9 gm. Within two hours the blood sugar fell to 148 mg. The blood pressure remained unchanged. With the usual breakfast (toast and coffee) and two hours previous to the ingestion of 225 gm. of glucose, 20 grains of thyroid extract were administered by mouth. The blood sugar at the end of one hour was 149 mg. per 100 c.c. The average for the patient as frequently determined was not more than 135. There was no glycosuria. The injection of 2 c.c. of 10 per cent. boiled anterior pituitary lobe extract produced no thermic response.

The Goetsch test gave a blood pressure rise of 15 mm. without glycosuria. During a two hour period there was an increase in white blood cells from 13,000 to 14,900 affecting the neutrophils. A 5 mg. dose of epinephrin resulted

in a leukocytic increase from 11,575 to 27,000 also neutrophilic, the blood pressure rose from 130 systolic to 175 systolic during the first hour and there were slight subjective symptoms of cardiac discomfort. The urine contained 3,375 gm. of sugar. The subcutaneous injection of 1 mg. of neutral sulphate of atropin produced moderate pupillary dilatation and accelerated the pulse from 100 to 115 over a period of several hours.

The administration of 0.01 gm. of nitrate of pilocarpin resulted in a scarcely perceptible supra-orbital sweating and possibly slight salivation.

The basal metabolism as determined by Dr. Jonas gave the following figures: respiratory quotient 0.85, total calories per hour 90.74, total calories per square meter per hour 53.7. The latter figure is 7 above the normal for a girl of 12, 14.5 above the normal for a person of her maturity.

Discussion.—The patient showed neither vagatonic nor sympathicotonic symptoms nor signs. She had, however, a syndrome of skeletal overgrowth, pathologic adiposity and unusually high sugar tolerance; the latter could be promptly decreased by the administration of pituitary extract but not so definitely by thyroid extract. Neither the character of the adiposity nor the appearance of the skin were suggestive of hypothyroidism and no other signs with the exception of the high glucose tolerance indicated the possibility of such a condition. The adiposity was more or less generalized. It had resulted in a recent increase in weight of 55 pounds and a present weight of 155 pounds, which was 82 pounds overweight for her age and 53 pounds overweight for her height. Referring to the Prudential Life Insurance Company tables, girls of her height and of an age, 3 to 7 years greater, weigh 35 pounds less. The highest normal weight for women of this height at any age is 14 pounds less. There is, however, a definite skeletal overgrowth. Aside from a 2 inch increase in height during a period of ten months, we have to consider certain skeletal measurements which compare in an interesting way with the normal for our patient's age. The ratio of the height of the symphysis to the total height is 54.8 per cent., being 4.8 per cent. greater than normal. The ratio for the umbilicus is 62.5 per cent., a figure which is not reached in any race. The length of the arm is 70.5 cm., 9 cm. above the normal and the ratio to the height is 42.2 per cent., 1.7 per cent. above the normal. The span is 2.25 cm. above the normal. The ratio of the span to the height is 105 per cent., 5 per cent. above the normal. There was, therefore quite definitely, in addition to a generalized overgrowth, a relative increase in the length of the long bones. If the pituitary disease had occurred later in life or after epiphyseal union, the result would have been the development of an acromegaly instead of gigantism.

Certain minor physical characteristics of some interest were the spacing of the teeth, the low temporal hair growth and the slightly tapering hands.

In connection with the negative roentgen-ray findings and other neighborhood signs, Cushing (*Am. J. Med. Sc.* **64**:313, 1913), and more recently Timme (*J. Nerv. & Ment. Dis.* **1**:460, 1919), have both called attention to the not infrequent occurrence of a normal sella in pituitary disease of a type similar to the one here presented.

The child had developed something which simulates gigantism and is succeeded by adiposity together with unusual sugar tolerance without neighborhood signs. It may be assumed that there was a hyperfunction of the anterior lobe, which, prior to epiphyseal ossification, resulted in skeletal overgrowth. The present posterior lobe deficiency completes the syndrome and accounts for the high sugar tolerance, the adiposity, the subnormal temperature, the somnolence and the dry skin.

These cases are important because they present a definite indication for treatment. The administration of the extract of the whole gland has been commenced with the intention of increasing the dose until the sugar tolerance is normal.

DISCUSSION

Dr. J. Hendrie Lloyd referred to an unfortunate experience with a case of dystrophia adiposo-genitalis in a man 22 years of age, who had attained a weight of 230 pounds, who had arrested sexual development, beginning optic neuritis and a larger sella turcica. Dr. Lloyd had him under observation for a few weeks. He was not quite so thoroughly studied as the patient of Drs. Strecker and Newcomer, but the glucose reaction did not show increased tolerance. He began to have curious epileptiform attacks and developed severe headache. The case seemed to be such an unmistakable one that Dr. Lloyd had him transferred to Dr. Frazier, who agreed with the diagnosis and decided to operate. The first incision was made—the bone had not been lifted—when the patient had symptoms of respiratory failure. He was kept alive for ten hours, with artificial respiration. Unfortunately, necropsy was not obtained. It was extraordinary that ether narcosis produced respiratory failure so quickly. As necropsy examination could not be made the diagnosis was not fully confirmed.

Dr. Francis X. Dercum said that in these remarkable cases of endocrine disorder it appeared that more than one gland is affected. Dr. Strecker had beautifully demonstrated his case to be one in which the pituitary gland was affected. Dr. Dercum questioned whether it was the only one; whether other glands did not react in some way. Sometimes a hint is given by the menstrual function.

A FORM OF BRACHIAL PLEXUS PARALYSIS CAUSED BY A BONY GROWTH OF THE UPPER THORAX RESEMBLING THAT CAUSED BY CERVICAL RIBS. Presented by Dr. William B. Cadwalader.

Dr. Cadwalader said that this condition did not appear to be generally known; similar cases do not seem to have been recorded.

In addition to the signs of irritation and compression of the trunks of the brachial plexus, there were also signs of compression of the blood vessels. The course of the disease has been slow, but gradually progressive. In Case 1 only slight progression had occurred in fifteen years, and in Case 2 there was no progression until after five years. The nature of the bony growth has not been determined, but its effects were striking and resembled those produced by cervical ribs. Unlike that condition, however, the pressure was exerted from below. The disease was diffuse, attacking more than one rib at a time, and the costal articulations as well. In Case 1 the disease was bilateral; in Case 2, unilateral. Syphilis was not present. It was significant that in Case 1 there was a clear history of four distinct attacks of pneumonia, and permanent evidences of an infectious process still remained in the lungs. Dr. Cadwalader believed that a chronic infectious process was the most probable cause of the bone disease in both cases.

Case 1.—*History.*—P. F., aged 55, admitted Nov. 25, 1919, complained of precordial pain which extended down the left arm.

In 1903 he first noticed weakness in his right hand. There was no pain. The weakness slowly progressed and extended, involving the muscles of the forearm and arm and then remained stationary. There was still considerable voluntary power. Since an attack of influenza a year ago, he had had precordial pain which radiated down the left arm into the hand and into the upper part of his chest. It was not increased by exertion but was occasionally sufficiently severe to keep him awake at night. He had had four attacks of pneumonia. For many years he had been exposed to cold and wet, and had used much alcohol.

Physical Examination.—Muscular development was rather poor. The right upper extremity was moderately wasted, particularly the muscles of the right hand and forearm. The muscles about the shoulder girdle were similarly affected, and to a lesser degree the corresponding muscles on the left side. The muscles of the left hand and forearm were normal. There was distinct weakness of the right upper limb, most marked in the distal portion but there was still considerable power. No paralysis was present on the left side. There were some fibrillary or fascicular contractions in the triceps of the right side. Sensation was normal. The biceps and triceps jerks on the right side were distinct, but less than on the left. The fingers of the right hand were held in slight extension at the first phalanx and partial flexion of the second and third phalanges. There was limitation of motion of the fingers. Flexion and extension were weak.

The lower extremities were entirely normal. The blood count was normal and blood Wassermann reaction was negative. Expansion of the chest was limited on the right side with increase of vocal fremitus and decreased resonance, especially over the upper lobe. At the right apex there was bronchial breathing and a few râles and at the right base a friction sound. The diaphragm on the right side was fixed; on the left it moved freely. There had probably been a chronic tuberculous process in the right upper lobe with retraction and probable cavity formation. The right basal pleura was thickened and the diaphragm was fixed there with compensatory emphysema on the left side. The heart was normal. There were varicose veins over the right upper thorax and shoulder, and on palpation the first rib and the right shoulder were rough and considerably thickened.

The roentgen-ray examination showed signs of an old tuberculous process of the upper lobe of the right lung with cavity formation and marked fibrosis pulling the arch and the trachea to the right with fixation of the outer aspect of the right diaphragm. The first rib on the right side and the third rib on the left side were greatly thickened. All other examinations were negative, except for marked infection of the mouth and teeth.

Case 2.—*History.*—C. R. E. In 1913, after using a wrench with his right hand, the entire upper limb became swollen. At the same time the hand and forearm felt numb and the fingers became strongly flexed on the palm of the hand, and the hand flexed on the forearm. He could readily straighten the fingers with the left hand, and they remained extended. After use, the right upper limb grew progressively weaker, and the swelling and discomfort continued, but after rest these symptoms entirely disappeared. The difficulty was greater when the limb was held extended over his head, as in hanging pictures. He had never had pain until a week before.

Physical Examination.—The right upper limb alone was affected. Grasp of the right hand was at first as good as the left, but became weak after continued effort. The biceps and triceps jerks were slightly exaggerated on

the right and normal on the left. Sensation everywhere was normal; there was no muscular wasting. There were marked varicose veins over the chest, particularly on the right side, and also in the upper portion of the right arm and shoulder. Recently he had had attacks in which he had difficulty in turning his head from left to right, associated with a feeling of faintness. Otherwise physical examination was negative.

A roentgen-ray examination showed premature ossification of the costochondral articulation of the second rib of the right side. At this point there was a fairly large osseous growth and ossification of the costochondral articulation of the first rib with some bony proliferation. External to this articulation the first and second ribs were fused together. The sternal end of the clavicle was slightly larger than its fellow, but the difference was not marked.

ON SOMATIC SYMPTOMS IN NERVOUS AND MENTAL DISEASES, WITH THE REPORT OF AN INTERESTING CASE. Presented by Dr. Francis X. Dercum.

Somatic or visceral phenomena in nervous and mental diseases present themselves under three different conditions. First, they may be directly due to and symptomatic of the underlying nervous affection. For example, in neurasthenia the deficient innervation and lessened inhibition present may give rise to characteristic phenomena presented by the circulatory apparatus, the digestive tract, the sexual apparatus or by various glands. In hysteria, the list of visceral phenomena that may be present is large: anorexia, vomiting, tachycardia, vasomotor phenomena, cough, rapid breathing, aphonia, polyuria, anuria, phantom tumor and especially local pains which lead to abdominal and other unnecessary operations. In a case presented some years ago, because of insistent complaint of pain, the left testicle, apparently entirely normal, had been removed. The pain, hypochondriac or, to speak more accurately, hallucinatory in character, at once transferred itself to the remaining testicle. Similarly much attention is at times given, especially in unrecognized cases of hypochondria, to the eyes, the nose, the throat, the digestive tract and other organs. I may mention a woman who had had thirteen pairs of eye glasses, twenty odd sets of false teeth, and who finally had had her stomach scrubbed out by the method of Dr. Turck, all to no purpose.

The second condition is one in which nervous symptoms are present but are secondary to visceral disease. For instance, general weakness sooner or later supervenes on visceral disease but this weakness differs radically in its symptomatology from that of neurasthenia, and is especially featured by the pains and other phenomena which direct attention to the structures involved or it may be to the general morbid process at work. Again, the exhaustion and the toxemia the result of somatic disease may in given instances give rise to mental disturbances, confusion, stupor or delirium.

A third condition encountered—but on the whole infrequently—is one in which primary nervous or mental disease and primary visceral disease coexist in the same patient; for example, brain tumor and hysteria, pelvic disease and hysteria. Here the situation may at times be misleading and difficult to unravel.

History.—H. S. T., aged 21, single, white, sailor, was admitted to the Jefferson Hospital, July 10, 1917, complaining of difficulty in swallowing. The family history was negative.

Personal History: The patient denied venereal infection and used neither alcohol nor tobacco. He had had an intermittent discharge from the right

ear in childhood, a convulsion at 6 years of age, and two more at 9 years. According to his unsatisfactory account, these began in the right index finger, and he was unconscious afterward for several hours.

In 1914, while at work, his sweater caught on the shaft of a donkey engine and he stated that he was turned around the shaft five times and sustained a 4 inch scalp wound over the left temporal region. His right ankle also was hurt.

Two years before admission he had two convulsions, one day apart. Each lasted about an hour and a half and was followed by many hours of unconsciousness; sometimes he said two, sometimes four. He said that he bit his tongue.

Present Illness.—One year ago while eating, he felt a contraction in his throat which he thought was the beginning of a convulsion. He went into the open air, but returned in a few minutes and finished his meal, though he did so with much effort. He had difficulty in swallowing and this difficulty in swallowing, he asserted, had persisted ever since. He stated that it was especially difficult to swallow solids and that swallowing was accompanied by much belching and often by regurgitation of food. He had lived for a long time mainly on milk and eggs. He was constipated and suffered from headaches.

Condition on Admission.—Esophagoscopy by Dr. Chevalier Jackson, disclosed a superficial erosion in the upper third of the esophagus contraindicating insertion of the tube beyond this point. Dr. Jackson suggested that a second esophagoscopy be done later.

Because of the occurrence of two convulsive attacks while chewing food at breakfast, the patient was afraid to masticate thinking it would bring on a convulsion. He had therefore been bolting his food and this was attended by a special effort which brought on some sensation of distress or possibly a spasm of the esophagus. The act was further followed by much regurgitation and belching.

Roentgen-ray examination was negative. Wassermann and other clinical examinations were negative.

Treatment and Course.—Under suggestion the patient was gradually persuaded to chew his food properly and he swallowed without apparent difficulty No convulsions were induced. Regurgitation and belching were also absent. The case was regarded as probably hysterical in origin, although other hysterical stigmas could not be elicited. The patient was later placed on ordinary house diet, ate normally and was apparently relieved. On July 23, 1917, he was discharged.

During his stay in the ward he was noted as rather peculiar. He was fault-finding and disagreeable; at times sullen and insubordinate; at times captious and complaining about his food. While these facts were noted at the time, no special emphasis was laid on them. No convulsions were observed at any time during his stay at the hospital.

He was readmitted on Oct. 27, 1917, again complaining of difficulty in swallowing. He stated that while chewing his food his throat felt as though it were swelling. He had again had special difficulty in swallowing solid food. The sensation of swelling in the throat passed away after taking a sip of water or after actually swallowing the food. He also complained of regurgitation.

Physical examination resulted negatively. Esophagoscopy by Dr. Jackson on Nov. 15, 1917, failed to reveal any erosion, stricture or spasm of the esophagus. As before, the patient was treated by suggestion and as before successfully. As before it was noted that he was a little peculiar. While he finally admitted that he no longer had difficulty in swallowing, he insisted that he had regurgitation. This, observation failed to confirm. It was now especially noted that he was reserved, uncommunicative, and suspicious in manner and introspective. He would ask the meaning of medical terms which he had heard used in connection with his case and was quite disturbed by the word "functional." It was noted also that he frequently busied himself writing on small pieces of paper, fragments of envelopes and other odds and ends. On one occasion with much solemnity he handed to one of my assistants an envelope, containing minute clippings of newspapers, a blade of a safety razor, the stump of a lead pencil and pieces of paper containing much closely written matter. The latter when examined consisted of a rambling and disconnected account of his symptoms and feelings in which a distinctly paranoid attitude of mind was discernible. He now also began talking of various strange sensations which he referred to the upper portion of his abdomen. His descriptions and the fact that the examinations of the abdomen resulted negatively indicated visceral hallucinations.

He left the hospital on December 7. His conduct continued to be peculiar. He was uncommunicative and solitary. He refused to eat the food provided by his aunt, and to which according to my inquiry there could have been no reasonable objection, but purchased his meals outside at various cheap restaurants. He offered no explanation, and it is not improbable that his conduct was the outcome of delusions, possibly having their origin in various visceral hallucinations. The latter he continued to refer to the throat and to the abdomen. Hallucinations of taste also appear to have been present.

Diagnosis.—The convulsive seizures which were neither reconcilable with those of hysteria nor of true epilepsy, remained unexplained. It was only the later appearing mental features which led to a correct appreciation of the case. Evidently the case was one of dementia praecox in an early stage, the symptoms of which at the time he first presented himself were just beginning to reveal themselves. The convulsions are to be regarded as among the epileptiform attacks — the motor crises — at times encountered in cases of dementia praecox, especially in the developmental period. The case teaches a valuable lesson as to the interpretation of local or visceral symptoms, when the latter have no or little physical foundation, and when, still further, hysteria offers an inadequate and unsatisfactory explanation.

PATHOLOGIC LYING IN A CRIMINAL. Presented by Dr. N. S. Yawger.

Dr. Yawger, after brief review of the meager literature, reported a case in which there was shown the marked character deviation of a pronounced and persistent fabrication, abnormal in type; this was a degenerate condition and was almost invariably accompanied by other forms of degeneracy.

In addition to the pathologic type of falsification the man was guilty of two attempts at homicide, one successful; three attempts at suicide, probably rape and possibly simulation of insanity. Subsequently, the convict was placed in the Pennsylvania Eastern State Penitentiary, where he is undergoing life-sentence.

The man, a young Lithuanian, while in the service of the Russian guard, became infatuated with a girl, whose father objected to the attachment because of a difference in religion. The man then attempted to kill both the girl and himself; they recovered. She fled to America but he followed, where, after ravishing her as she charged, he later murdered her.

Dr. Yawger remarked that the prisoner was without positive anatomic stigmas. In appearance, he was tall, erect and finely proportioned; his head was well shaped and his countenance was rather inviting; he was polite, quick-witted, of the utmost assurance, an incessant talker and with a great deal of general but superficial knowledge which he used with considerable linguistic ability.

As to his fabrications, his most remarkable invention was while awaiting trial in Moyamensing prison. It related to another atrocious murder in which the prisoner said he was implicated and which he attempted to tell a priest but later refused to confess him until the authorities had been apprised of the crime. He then reported that he had stood by while one man murdered another, that he saw the grave dug and the body placed therein. A detective was detailed to investigate the case but found that the presence of water rendered it a physical impossibility to have dug a grave there and it was concluded that the prisoner was simply "exercising his imagination." He still recalled the circumstances and regarded it as a good joke.

The prisoner was suspicious and was constantly making accusations against overseers and cellmates. He frequently reported that cellmates were secreting in their cells coffee, cocoa, sugar and money, all of which were contraband. He once charged that another prisoner was going to stab him with a pair of shears and at another time that two convicts were trying to cut through their cell door. With grave concern, he once reported that an inmate was hiding tools in a sewer, preparatory to an attempt at escape; on investigation, it was found that the cover to the sewer was securely cemented and had been so for years.

When pressed for an explanation he stated that "I was bluffing;" that "others have lied to me first;" that "often I lied to defend or to protect myself which I considered a justifiable fraud;" and that "sometimes I have lied to develop a situation about which I was suspicious."

The mental classification of the man is psychopathic personality. The outstanding features are: emotional instability, abnormal sexuality, pathologic lying, possibly still suicidal.

THE CURATIVE INFLUENCE OF INFLUENZA ON A CASE OF SPECIFIC MENINGO-MYELITIS WITH CYSTITIS. Presented by Dr. C. W. Burr.

This paper was published in the May number of the Archives of Neurology and Psychiatry, page 536.

DISCUSSION

Dr. Alfred Gordon said that about a year and a half ago he was called to see a case of paraplegia of long standing with much pain and bladder incontinence. The history suggested syphilis and the patient was given ten injections of neo-arsphenamin. The bladder disturbance improved and she began to move her limbs slightly. Suddenly she developed bronchopneumonia of great violence; the temperature reached 104 degrees. When she recovered from the bronchopneumonia, Dr. Gordon noticed immense improvement in the condition of the limbs, and at present the patient can walk a little.

Dr. William B. Calwalader said that cystitis retarding the return of power in paraplegic cases had been noticeable in cases of paralysis from gunshot wounds of the spinal cord observed in France. The work of Head and Riddock and others had demonstrated that return of function was almost nil unless cystitis could be cured. The importance, therefore, of the utmost care in the management of the paralyzed bladder cannot be exaggerated. Dr. Cadwalader also said that return of function after the administration of antisyphilitic treatment can be long delayed; function may reappear long after treatment has been stopped. Dr. Spiller had reported such cases.

Dr. Dercum thought that all were familiar with the results of infection in paresis. Pilcz of Vienna had brought about striking remissions by inoculating the patient with the virus of erysipelas. He thought Dr. Burr's case was unique.

Dr. C. W. Burr said there was one point he might not have emphasized sufficiently; namely, for six months after the woman left the hospital she received no antisyphilitis treatment of any kind. He was quite convinced that in paraplegics from all causes the cystitis was certainly a measure of loss of power. This was the only case of this kind he had ever seen, but he was convinced that if a patient had severe cystitis and that cystitis improved tremendously there would be some improvement in the legs. Dr. Burr said he had seen this result a number of times.

MONOPLEGIA SPINALIS SPASTICA. Presented by Dr. William G. Spiller.

This paper appears in full in this issue.

DISCUSSION

Dr. Charles S. Potts reported the following case: A man, 48 years of age, for the last two years had had purulent bronchitis, no tuberculosis. About six weeks ago he developed pain and weakness in the left leg. He now had some atrophy of the muscles of the thigh and distinct pain and tenderness in the left knee. The pain extended up the leg toward the hip. He had some fibrillary twitchings in the thigh muscles. Electrical reactions were normal. There was marked increase of the knee jerks, more so on the left, and ankle clonus on both sides. At times he had some fever. The sphincters were normal and roentgen-ray findings were negative. Dr. Potts said the man probably had a toxemia of some sort, possibly due to the purulent bronchitis and that he might have arthritis in spite of the absence of swelling and roentgen-ray findings. Possibly it may be a case like Dr. Spiller's, in which the roentgen ray failed at first to reveal anything.

Dr. Woods said that probably most of those present knew of the existence of these commissural fibers, which, according to Obersteiner, pass over as a regular constituent of every motor root, spinal and cerebral, and thus bring the muscles corresponding to each root under the influence of the motor cells of the same level in the opposite side; but that the Society was indebted to Dr. Spiller for showing this example of the probable functioning of such fibers. If a particular muscle receives its chief innervation from a certain segment, it is believed that after the destruction of the cells of that anterior horn other cells in neighboring segments above and below on the same side may assume control of the muscle. Dr. Woods understood from Dr. Spiller

that in this patient the lesion had extended so far in a vertical direction as to remove this possibility and therefore he could see no other explanation of the existing spasticity except the one Dr. Spiller had proposed.

PRESIDENT'S ANNUAL ADDRESS. Delivered by Dr. J. Hendrie Lloyd. This paper will be published in full in an early issue.

NEW YORK NEUROLOGICAL SOCIETY

Three Hundred and Seventy-Ninth Regular Meeting, March 2, 1920

Walter Timme, M.D., *President*

PRESENTATION OF CLINICAL MATERIAL. CHRONIC NONDEGENERATIVE HEREDITARY CHOREA. Presented by Dr. I. S. Wechsler.

This patient showed a clinical picture closely resembling Huntington's disease, but certain distinctive features suggested that it might be a distinct clinical entity. A married woman, aged 36, had had peculiar movements of arms, hands, body and legs and twitchings of the face for about sixteen years, gradually increasing in intensity for a time, then remaining comparatively unprogressive. A slight weakness of the heart and faintness were complained of at the onset, which is said to have followed a miscarriage. There were no convulsions, biting of the tongue, amnesia, etc. The attacks were closely linked with the patient's emotional state, suggesting a possible hysterical condition. The patient's father, who was the uncle of her mother, also suffered from chorea for twenty years. One brother had shakings. Of her children, one daughter was not nervous, but had poor eyesight and nystagmus; the second daughter had twitching and attacks of weakness. The shaking was not choreic. Two small boys both had chorea and nystagmus.

The patient showed a number of abnormal involuntary, irregular, jerky purposeless movements of whole parts—arms, legs, body, hands, and twisting of the whole body. The eyeballs wandered in irregular fashion. All these movements were intensified by emotion while control inhibited them for only a short time. The movements were in general more rapid than in chorea. No pathologic reflexes were found. Vision and hearing were normal, except for the choreic nonrhythmic movement of the eyes. Mental status was perfectly normal, a slight tendency to forgetting probably being due to lack of attention.

The oldest boy, aged 9½, at 7 years developed a condition diagnosed as acute chorea. The condition improved somewhat after six months. At 7½ years there was a second acute attack which still continues.

The younger boy, aged 6, has had slight twitchings since he was 3 years old. Slight unsteadiness in equilibratory and nonequilibratory tests, of a choreic nature, was found on examination. Some nystagmus on looking forward and trying to fix the gaze was noted.

Unlike Huntington's chorea, the onset of the attack was at the early age of 20. The movements are quicker, the face shows more grimaces, speech is differently affected, somewhat forced and slow but not scanning. The gait is clownish. Mental degeneration is absent. Hysteria might be adduced as a cause, especially hysteria associated with chorea, while other forms of chronic

chorea, such as chorea gravidarum and paramyoclonus multiplex, have some features suggestive of this case, but do not correspond sufficiently to warrant the diagnosis. The point of particular interest is that it is a nondegenerative, nonprogressive type of hereditary chorea.

DISCUSSION

Dr. Sylvester R. Leahey of Brooklyn stated that he had seen conditions similar to that of Dr. Wechsler's patient in women of about 50. These had paranoid and suicidal tendencies. In view of the youth of the patient, he thought that the mental symptoms that could not be observed now, might develop later.

Dr. J. H. Leiner did not believe that this case came under the true category of Huntington's chorea, since the choreiform movements were entirely too lightning-like in character.

HYPERTHYROIDISM IN A GIRL OF NINE YEARS OF AGE. Presented by Dr. Morris H. Frantz (by invitation).

This case was of interest because of the infrequency of the condition in children. When the patient came to the Neurological Institute Clinic, she was fidgety, would develop rages and had palpitation on violent exercise. Muscular sthenia, ocular manifestations and a distinct exophthalmus were present. Tachycardia and slight tremor of the hand were also noted. The mental age was 12½ years.

The patient's father had rheumatic arthritis, the mother suffers from hyperthyroidism. Goiter was present in a maternal aunt. The child was born in a town in Germany in which goiter was prevalent. The mother developed goiter at the time of the child's birth. The same condition was diagnosed in the child when 1½ years old. The condition became aggravated at the time of the emigration of the family to America during the submarine blockade.

DISCUSSION

Dr. Walter Timme called attention to other glandular stigmas present, and questioned whether the case could be called one of hyperthyroidism since hypothyroidism and hyperthyroidism and hypoadrenalism all appeared to have been present at some time. The absence of lateral incisors, and the abnormal configuration of the teeth were of particular interest for the glandular study.

Dr. J. H. Leiner asked whether this case might not be considered a fruste type since the patient was well preserved and did not show any of the cardinal symptoms of an advanced thyrotoxicosis.

Dr. Frantz said that hypothyroidism and other pluriglandular syndromes had been apparent in the patient, but that since the hyperthyroidism was the prominent feature he had placed special emphasis on that condition.

ACUTE INFECTIOUS MYOCLONUS MULTIPLEX AND EPIDEMIC MYOCLONUS MULTIPLEX. Presented by Dr. J. Ramsay Hunt.

Dr. Hunt called attention to the problem of localization of acute infections in some part of the nervous system. The varieties of clinical types in Heine-Médin's disease, for instance, emphasized that certain strains of the same infective organism might have special affinities for certain tissues of the nervous system, and thus bring about the special clinical type of reaction.

Such special forms of localization of acute infection are to be found in acute infectious myoclonus multiplex and epidemic myoclonus multiplex. The form is characterized by lancinating pains, muscular contractions and twitchings and a delirium of toxic origin. This group of symptoms constituted a well defined clinical type of neural infection which differed from those previously recognized and was encountered both in sporadic and epidemic form.

The onset of the disease is acute and is characterized by shooting pains of great intensity in the trunk and extremities. Spinal pains are sometimes present. The pains are followed by characteristic muscle jerks, waves and twitchings (myoclonus multiplex, myokymia and fibrillary contractions). The contractions make their appearance first in the parts in which the pains were first felt. A week may elapse in some cases between the appearance of the pains and the myoclonus and myokymia. The twitchings are bilateral, multiple and may become generalized. There is sometimes a tendency to localization in certain regions of the body, especially in the abdominal musculature. The contractions are quick and of short duration, individual muscles or portions of muscles are involved, but not synergic groups. Slight movements of the toes, fingers and extremities may occur in severe myoclonic twitchings, but never to the extent found in chorea or cortical myoclonia.

There is usually a moderate fever. In some cases which proved fatal the temperature rose in the later stage of the disease. An acceleration of the pulse rate was noted, and in most cases, a delirium which varied in duration and intensity with the degree of infection. There is often marked hyperidrosis, and the degree of sweating seems to bear some relation to the activity of the myoclonus phenomena. There is no paralysis or paresis of any muscle or group of muscles; no anesthesia is encountered, with the exception of occasional transient areas of hypalgesia. No ataxia and no loss of deep sensibility; tendon reflexes are present and active. Rarely the knee jerks may be diminished and the Achilles jerks are absent during the height of the disease. The cranial nerves show no evidences of involvement except for the myokymic twitchings. The optic nerves are normal. Skin reflexes are present and equal (no Babinski sign). When abdominal myokymia was present, the abdominal reflexes were exaggerated.

Dr. Hunt had observed twelve cases of this type in sixteen years; two cases were seen more than ten years ago, and the remaining ten within the last three months. The first cases were evidently sporadic, the latter epidemic. The distinguishing features, acute pain of lancinating variety, with muscular waves and twitchings were always present. Delirium was present in eight of the cases. The myoclonus delirium was a characteristic toxic delirium with hallucinations, illusions and transitory delusions. Restlessness, insomnia, apprehension, disconnected thought and mental confusion were present. Apathy and a tendency to stupor were sometimes encountered in the late stage. In the four cases without distinct delirium there was insomnia, restlessness, irritability and excitement in the early stage and later a tendency to apathy and dulness.

That the disease is infectious is evident from the character of the onset, the fever and delirium. Multiple neuritis and acute poliomyelitis may be excluded as diagnoses since the paralysis or weakness of the muscles associated with these diseases is absent. There is no appreciable tenderness along nerve trunks. Dubini's disease may also be excluded since it involves paralysis. Epidemic lethargic encephalitis is an especially interesting possibility.

The epidemic myoclonus multiplex probably belongs to this group and represents a special myoclonus type of this affection. The infectious agent of epidemic encephalitis and epidemic myoclonus multiplex is apparently the same.

The motor and sensory symptoms of the disease are only irritative in character, in spite of the severe and sometimes lethal infection of the nervous system. There is no paralysis or anesthesia, and this fact gives the disease an added interest, since the myoclonus syndrome is not found in other forms of spinal and neural infections.

DISCUSSION

Dr. Charles Rosenbeck asked whether any of the patients with myoclonic phenomena had subsequently developed lethargic encephalitis. In three cases, which he had observed, the violent neuralgia, myoclonus, mild temperature and mental confusion were the dominating symptoms. There were no demonstrable sensory changes except dysesthesia. One of these patients recovered in a short time, without further incident. The others, however, after a variable time, gradually became merged into the syndrome of lethargic encephalitis.

Dr. C. C. Beling of Newark reported that he had seen five or six cases of similar sudden onset of pain, occupational delirium and muscular incoordination.

Dr. William M. Leszynsky had seen several cases of encephalitis in which there had been clonic spasm of the abdominal muscles. Another patient developed a clonic spasm of the diaphragm without hiccup.

Dr. M. Neustaedter reported three cases, seen within the last four weeks, of undoubted lethargic encephalitis complicated with myoclonus involving the abdominal muscles in two cases and the facial muscles on the left side of the face in one.

Dr. Gregory Stragnell (by invitation) asked whether hiccup was a feature of the symptomatology observed by Dr. Hunt. From Canada it was reported that 50 per cent. of the persons suffering from encephalitis had a hiccup that persisted for seven or eight days.

Dr. J. H. Leiner had had a case with diplopia. The diplopia developed ten days after the beginning of the disease. It was diagnosed as lethargic encephalitis. This patient had choreiform movements mostly of the upper extremity, accompanied by chewing and swallowing movements of the mouth. He also showed right-sided abdominal movements. Additional interesting features were the occurrence of an additional diplopia (objects appearing one above the other) after the initial diplopia had disappeared, in which the objects appeared from side to side. Again an Oppenheim and Gordon reflex appeared four weeks after the onset of the disease and also a distinct line of hyperalgesia a little below the mammary line in front and about the seventh cervical behind, together with a most distressing burning sensation in the fingers of the right hand, limited to the ulnar distribution. A Sergent's white line was persistent, showing a hypo-adrenia.

Dr. Hunt said that hiccup had not been a feature of the clinical symptoms observed by him. Abdominal waves were present, but the diaphragm was not affected. There was no evidence of organic disease of the brain other than delirium. He had, however, observed combination forms of epidemic encephalitis and myoclonus multiplex.

MEMORIAL RESOLUTIONS ON THE DEATH OF DR. E. E. SOUTHARD. J. RAMSAY HUNT.

Mr. President: It is fitting that the members of this Society should pause in their deliberations and give expression to the loss we have sustained in the death of Dr. Southard. The news of his fatal illness in this city on February 8 touched us with a peculiar poignancy, as it came only a few days after his visit here as our guest. On that occasion he gave us one of those delightful evenings for which he was so justly famous, in which the subject of psychiatry was enlivened by his rare wit and philosophical mind. The memory of this occasion will always be treasured by those of us who heard him, for he was in one of his happiest moods. There are, I venture to say, but few other men in American medicine today who combine all the intellectual and personal gifts which made that address possible.

Elmer Ernest Southard was born in Boston in 1876, and at the time of his death was 43 years of age. He was a graduate of Harvard and of the Harvard Medical School, and subsequently became closely identified with his alma mater as Bullard Professor of Neuropathology and Assistant Professor of Psychology. At first a neuropathologist, he soon manifested an interest in psychiatry, and in 1912 became director of the Boston Psychopathic Hospital. This institution, under his leadership, grew to be one of the great centers for students of psychiatry in this country and Southard himself became a figure of national importance. He was an active and many-sided man. He held the directorship of the Eugenics Record office at Cold Spring Harbor, and was also a scientific director of the Psychological Laboratory at Bedford Hills. It was not, however, this array of appointments and honors that made Southard a unique and growing force in medicine, but rather his personality and philosophical type of mind.

A disciple of William James and Josiah Royce, he brought to medicine a mind which had been steeped and trained in the philosophical method. This, with his other gifts, made him one of the leaders of his generation.

While in his later years he became a man of affairs and an organizer of repute, he never lost the simple ideals and habits of the scholar, and this was perhaps one of his strongest characteristics, and contributed so largely to his unique position in medicine.

One can measure, in a way, what Southard had already accomplished in his life's work, but like all great men he was in the making and who can foretell what large accomplishments lay ahead of him? If one contemplates for a moment the loss to medicine if Sir William Osler had been struck down in early manhood while still teaching the Institutes of Medicine and McGill University, one can better gage what American psychiatry has lost with the passing of E. E. Southard.

TWO CASES OF BRAIN TUMOR. WITH SPECIMENS AND LANTERN SLIDE DEMONSTRATION. Presented by DRS. C. C. BELING, H. W. MARTLAND (by invitation) and W. B. EAGLETON (by invitation).

DR. BELING: CASE 1.—A man aged 25, an experimental engineer, suffered superficial burns of both corneas in an explosion of barium chlorate in 1913. Recovery was complete. In October, 1918, he began to see double, and lenses and general treatment failed to produce improvement. In March, 1919, an examination by Dr. Eagleton showed: vision in the right eye 20/100, in the left eye 20/50; marked papillitis of the right optic nerve; diplopia as a result

of paralysis of the superior rectus of the right eye and a spontaneous nystagmus. There was increasing difficulty in looking upward. When Dr. Beling examined the patient the papillitis of the right optic nerve was marked. There was no deviation of the tongue or tremor. Knee jerks and plantar reflexes were normal except for a slight tendency to slow reaction on the right side in the latter. On May 15 the patient developed influenza and has been ill since that time. Since July 1 a dull pain on the top of his head with slight frontal headaches has persisted. His mental condition has apparently deteriorated. He was often nauseated and vomited. He could walk for a short time, then his body would stiffen and his head jerk back. An operation, a left subtemporal decompression, was performed by Dr. Eagleton. The brain was under great tension, the dura widely exposed. The operation was followed by an uninterrupted recovery, and the man's condition improved, although the papilledema persisted. Several weeks later, however, greatly increased intracranial pressure was apparent. For the first time he showed a tendency to fall backward. Examination at this time showed an intense double papilledema. There was generalized tremor and profuse hyperhidrosis and tonic contraction of the muscles. Knee reflexes were exaggerated. Clonus of the toes was found, the mental state was somewhat confused; the patient could not remember dates, but knew the year and that he was in the hospital. Cerebration was difficult and tremor was produced by attempts to answer questions. Priapism was noted; no abnormal psychosexual phenomena were present. His condition became steadily worse, the tremor increased, eyes bulged, jaws were set. On November 8 he began to have convulsions in rapid succession, profuse perspiration, and began to grow cyanotic. He died a few hours later.

Slides of the hospital history and the necropsy findings were shown by Dr. Martland. The diagnosis had been tumor of the midbrain. It was found, however, that there was a small psammoma of the pineal gland. An enormous dilatation of the third ventricle had resulted. The pineal gland was visible in the roentgenogram. Dr. Eagleton, in discussing the surgical features of this case, pointed out how useless further decompression would have been. The possibility of a pineal gland tumor had never been suggested, since the usual headache was lacking. Relief for the increased intracranial pressure was sought by the left sided decompression earlier in the case, since Dr. Eagleton had come to the conclusion that the patient was left handed; but this had had no effect on the papilledema.

Case 2.—A man aged 40 years, began to lose weight, vomited every morning, had increasing dizziness and began to see double. There were severe headaches. Examination by Dr. Beling showed slight swelling of the right papilla, vertigo, ataxia and asynergia of the right side. There was deviation to the right on walking, and nystagmus with rapid movements to the left and slow to the right. Hearing was about equal. Symptoms pointed to a lesion in the posterior chamber and subtentorial pressure. There was probably a cerebellar tumor with slight involvement of the pons since the left side of the face and hearing showed slight disturbance.

Dr. Eagleton noted the following phenomena: Romberg sign; spontaneous pointing deviation of the right hand to the right; more marked spontaneous nystagmus on looking to the right. Rotation to the right and sometimes to the left produced nystagmus, though whether spontaneous or induced could not be determined. Duration apparently was about eighteen seconds. Rotation to the left produced nystagmus of fifteen seconds' duration. There was

deviation of both hands to the left. No dizziness appeared in either rotation. Cold caloric stimulation in the ears produced no nystagmus, no past pointing or dizziness. Nystagmus could be induced by turning the head backward, showing that tracts of the vertical canals were not functioning while the horizontal were functioning.

The tumor was thought to be pressing somewhat on both sides. Cases of this sort, Dr. Eagleton felt, where the exact nature of the tumor was not recognized, were better not operated on.

DISCUSSION

Dr. J. W. Stephenson said that in his opinion the primary operation for any brain tumor should be puncture of the corpus callosum, especially puncture for midbrain tumors with which dilatation of the ventricles is most probable. Where large quantities of fluid are evacuated he advocated no further operation. In the absence of evacuation of fluid decompression was indicated. In tumors in locations other than the midbrain he would advise therapeutic lumbar puncture, particularly indicated in frontal tumors. He cited three cases of frontal tumors relieved of symptoms by lumbar puncture. In time the symptoms reappeared but lumbar puncture again relieved. In one case seven punctures were performed at intervals of seven to ten days, the puncture being dependent on return of symptoms. Each puncture gave decided relief.

Dr. Alfred Taylor advised wide removal of bone and the extensive opening of the dura in decompression cases, especially when the operation was on the left side as the speech center is more likely to be affected by limited work over it. In cases of high pressure ventricles should be aspirated. A needle inserted into the ventricle to evacuate the fluid was an assistance before opening the dura.

Dr. E. D. Friedman mentioned the fact that the first patient showed an inability to rotate the eyes upward. This sign is very suggestive of lesions in the midbrain, since the more cephalad portion of the posterior longitudinal bundle controls the vertical movement of the eyes. This sign has been present in many of the cases of encephalitis seen in the present epidemic. Here, too, the peri-aqueductal localization of the encephalitis process was shown by this inability to rotate the eyes upward, together with oculomotor paralyses. Since this patient had already reached maturity, symptoms from involvement of the pineal body were absent. In younger persons lesions in this area involving the pineal gland are associated with sexual precosity and the premature development of the secondary sex characters. In one patient there were noted, with the other evidences of a lesion in this area, peculiar convulsive seizures, with athetoid movements in the hand, without loss of consciousness. This was attributed to involvement of the nucleus ruber which lies in the tegmentum of the brain stem.

Dr. Friedman asked, with reference to the second case, whether there was any conneal anesthesia on the side of the tumor. Oppenheim mentions this sign as being extremely diagnostic of posterior fossa neoplasm. It is homolateral to the tumor and said to be due to lesions of the spinal fibers of the trigeminus which in their ascent lie near the periphery.

Dr. Eagleton agreed with the advisability of wide opening of the dura for decompression when the case demanded, but emphasized again the usual danger to the speech center, and the uselessness of decompression in cases like the first, or cases that were as far advanced as the second.

TRAUMA AND OTHER NONSYPHILITIC INFLUENCES IN PARESIS.

Presented by Dr. Michael Osnato.

Literature yielded practically nothing on the influence of trauma in the production or precipitation of neurosyphilis. From the few cases which could be included under this category at the Vanderbilt Clinic in the past three or four years, from Dr. Osnato's own files only thirteen cases could be assembled. These cases were all proved either by laboratory examinations or necropsy. This deficiency of cases in which trauma was an associated factor, whether recognized or not, is to be noted in Southard and Solomon "Case History Series" in which a few cases only are mentioned. The post-traumatic paresis usually occurs, these authors state, citing Mott's study of the same subject, after at least a week's interval, since the time required for the destruction in the brain productive of the necessary symptoms would seem to be at least that long. Three months was the limit of time that Southard felt should be set to determine the influence of trauma as a causative factor. An increase in the number of cases of neurosyphilis during the war noted by the Canadian medical officers is thought to have been due to the great strain at the front, and the frequent physical injuries resultant on being buried.

A possible influence in the production or stimulation of paresis is accordingly granted by other observers. Dr. Osnato described the following cases in support of the traumatic theory: A patient who had been struck on the back of the head eighteen months before by a heavy object soon developed mental inefficiency. The only mental signs at present are perseveration of thought and speech and memory defect. The physical signs of paresis are present. A second patient, in whom the trauma was emotional, had been entirely efficient and dependable in his work until he was drafted into the army. His mental reaction was like a war neurosis in every respect. After diagnosis as a psychoneurotic and after his discharge he continued to fail and finally came to the clinic complaining of gross memory defects, fifteen-minute attacks of amnesia, dulness and retardation amounting almost to negativism and loss of interest. The mental picture was that of a psychoneurosis of the phobic type; a diagnosis of general paresis was made from investigation of the blood and spinal fluid. The problem of the emotional factors in the production of this condition is forcibly introduced here. The study must be speculative since the exact physiologic changes that may take place as a result of fear or other emotions is not known. A third patient developed the paretic picture after a prolonged etherization. Following an operation she complained of pains in the chest, legs, abdomen and right upper extremity. Grave memory defects also appeared. The physical signs of tabes were present, but mentally the patient was a general paretic. She is now under treatment and shows progressive mental deterioration, however, without delusions or hallucinations. The fourth patient had a severe attack of influenza and complained of lancinating pains in the right arm and both legs shortly after. She became depressed, slept badly, had tremor of the face, hands and tongue, was ataxic and had a moderate memory defect. The blood Wassermann reaction and spinal fluid findings in this case were those of a cerebrospinal syphilis rather than general paresis. Before the attack of influenza she had been perfectly well. In the last patient cited the trauma had been caused by a falling plank which struck the right parietal skull and glanced off striking the dorsal region of the spine. He was in Bellevue Hospital three days. There was evidence of a depressed fracture of the right vault of the skull in the frontoparietal region, over the Rolandic area. Left hemi-

plegia had developed when he left the hospital. A few days later there was unsteadiness of gait, ataxia, Romberg, typical paretic speech, stuttering, memory defect and tremor. The initial hemiplegia was undoubtedly due to the trauma. Up to the date of his injury he had worked steadily, and had shown no apparent signs of paresis.

Dr. Osnato emphasized that there are undoubted acute and chronic pathologic lesions of the brain ascribable to trauma of the head. Something seems to alter the permeability of the blood vessels of the brain, thus making possible the attack of the spirochetes on the brain tissue. In the cases described craniocerebral injury seems to have precipitated paresis. The toxins of influenza, infections or ether may have an effect similar to trauma, while the effect of emotional stress offers material for interesting investigation.

Dr. S. R. Leahy recalled Dr. Kraepelin's statement that paretic symptoms make their appearance immediately or shortly after a head injury. Sometimes the accident results from the paretic unsteadiness or occurs during a seizure. Whether in head injury, as is often assumed, one has to reckon with a circumstance which favors the outbreak of paresis, cannot, in the present state of our knowledge, be either proved or argued. Macfie Campbell, in his extensive works on paresis, states that even microscopically one cannot always tell which lesions have resulted from injury and which from paresis. Until we have more pathologic evidence the questions of the influence of injury in paresis must remain unsettled.

Dr. Osnato again pointed out that the patients had been efficient before injury, and then developed general paresis. Aside from the acute signs of trauma in the brain, other late signs, such as gliosis, nerve cell sclerosis, which recalled specifically the paretic brains, were to be found on necropsy. The same findings might be noted in traumatic insanity.

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Book Reviews

DIE HYSTERIE ODER SOGENANNTE PSYCHOGENE NEUROSE: Eine psychopathologische Studie auf dem Grenzgebiete des Nerven-und Seelenlebens. By OSCAR LESSING, Dr. phil. et med., Arzt in Berlin. Berlin, 1920, S. Karger, p. 45.

This is an educational essay for the laity presenting generally accepted conceptions of the nature and symptoms of hysteria. Because of the enormous increase in the number of cases of hysteria caused by the great war, the author thinks that the people at large should know more of this nervous disorder. The diction is diffuse, but readable, and the matter is sound.

TRAUMA UND NERVENSYSTEM: Mit gleichmässiger Berucksichtigung der Kriegserfahrungen der Ententeländer und der Mittelmächte. By DR. TH. VAN SCHELVEN, Neurolge der Niederlandischen Sanitätsmissionen. Berlin, 1919. Verlag Von S. Karker, p. 303.

This work is a systematic presentation of our knowledge of traumatic injury of the brain, cord and peripheral nerves with a brief (twenty-four pages) consideration of traumatic neuroses. The practical basis of the book is the author's war experience. As a neutral he had the advantage of observation on both sides of the line, and he avows an effort to present the sum of international knowledge of trauma of the nervous system acquired during the war. As a summary the book is valuable but it lacks the punch that is given by an author who is himself master of the subject. That the literature has been exhaustively consulted is evident, but too often the name of an author is given without the bibliographic reference. For instance, on page 3 authors are referred to twenty-two times with only six indications of the source in the literature. Many words are wasted in statements of the obvious. One scarcely needs to be told that "long continued compression, which need not be very intense, may cause nerve paralysis," and that "gunshot injuries of the skull belong to the most dangerous wounds of war and peace." All of this gives the work a pedantic air and the reader the impression that the author either is not quite sure of himself or is a beginner in book writing.

Although war work apparently is responsible for the book's having been written, it is in no sense to be regarded as a work on war pathology and practice, but it is an attempt to cover peace trauma quite as completely. The entire subject is treated in the most methodical way. The section on peripheral nerves (107 pages) takes up in succession pathogenesis, pathologic anatomy, clinical manifestations, diagnosis, therapy and prognosis. Under diagnosis there are nine subheads. The same painstaking method is pursued throughout so that the student is sure to find every phase of the subject considered. The work contains a great amount of information and summarizes a vast deal of knowledge. What is lacking is discriminating emphasis. The book is an honest, solid product of considerable value, but the tyro will have difficulty in separating the essential from the nonessential and the ordinary from the exceptional. The experienced neurologist or surgeon will find much that is superfluous.

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